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STUDIES
IN
HUMAN BIOLOGY

BY

RAYMOND PEARL

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TO

THE MEMORY

OF

A GREAT HUMAN BIOLOGIST

WILLIAM STEWART HALSTED

PREFACE

The material brought together in this book represents what has been a major interest of the writer's scientific life for more than twenty years past. Of all the animals that people the earth man is, to himself, the most interesting. Twenty years ago it was generally thought unseemly or worse for a professional biologist to concern himself scientifically with man. His material was held to include all living things except man, who belonged to the anthropologist. Now this situation is a good deal altered, and quite certainly for the better. Anthropologists know more now than they did then about general biology, and are employing usefully the principles of that branch of science in their own work. Biologists are at the present time in no way likely to suffer either social or economic ostracism if they venture to study human problems.

In some part, and perhaps a fairly large one, this encouraging bit of intellectual evolution has been aided by the concurrent development of biometry. The workers with this technique have usually been biologists in the beginning, and in some cases at any rate have managed to retain a firm hold upon this solid anchor of biometric sanity. At the same time it has been obvious to anyone that nowhere could the biometric method be applied with greater prospect of profit than to human problems. Hence we have seen biometry acting in some sense as a *liaison* officer between general biology and anthropology.

The major portion of this book has appeared before in the form of papers in various scientific journals. Many of them have long since been unavailable in separate form. There has been for some time a demand for their reprinting, and the increasing number of students in the Department of Biometry and Vital Statistics of the School of Hygiene has tended to make this demand rather insistent. Chapters IX, XI, and XXV embody material here published for the first time. All of the old material has been carefully edited, and in some cases extensively revised.

A book of this sort can make only such claim for unity as inheres in the *point of view* of its author. But after all is not a definite and fairly consistent point of view perhaps the most significant kind of unity in a scientific book? It is this thing, more than any other, which creates the soul of such a book. If this view of the matter is accepted in any degree the present work will gain rather complete absolution for the rhetorical sin

under discussion. Every page reflects, in the most highly unified manner, the scientific personality of its author. Whether the results of this fact are thought to be good or bad, it is a fact, and one about which nothing can be done at this late date.

To the following journals and persons the author is indebted for permission to reprint material appearing in this book: *Biometrika* (Professor Karl Pearson); *Journal of Comparative Neurology and Psychology* (Dr. Milton J. Greenman); *American Anthropologist* (Dr. A. V. Kidder, Secretary-Treasurer); *Biological Bulletin* (Dr. Frank R. Lillie); *American Naturalist, Science* (Dr. J. McKeen Cattell); *American Journal of Hygiene* (Dr. C. E. Simon); *American Review of Tuberculosis* (Dr. Allen K. Krause); *American Philosophical Society* (Miss Rebecca E. Kirkpatrick, Assistant Secretary); The W. B. Saunders Company (Mr. R. W. Greene, Vice-President); *Journal of Industrial and Engineering Chemistry* (Dr. H. E. Howe); *Proceedings of the National Academy of Sciences* (Dr. E. B. Wilson); Public Health Reports (Surgeon-General Hugh S. Cumming); *Journal of the American Statistical Association* (Professor W. F. Ogburn); *Johns Hopkins Hospital Bulletin* (Dr. Winford Smith); *American Journal of Public Health* (Dr. Homer N. Calver, General Secretary); *Geographical Review* (American Geographical Society, Dr. Isaiah Bowman); *Proceedings of the Washington Academy of Sciences* (Dr. Arthur L. Day); *Metron* (Professor Corrado Gini). All of these institutions and persons I wish to thank most heartily for their kindness.

To Dr. John Rice Miner and Miss Helen Trybulowski I wish to express my grateful appreciation for aid in proof-reading and in other ways in the preparation of this book.

RAYMOND PEARL.

TABLE OF CONTENTS

PAGE

Preface 5

PART I

Considering Man as an Animal

CHAPTER

I. The Weight of the Human Brain 15
II. Intelligence and the Size of the Head 93
III. Race Crossing and the Sex Ratio 103
IV. The Sex Ratio Among the Jews 114

PART II

Biological Aspects of Vital Statistics

V. Congenital Malformations 123
VI. Centering Infant Mortality 132
VII. Mortality and Evolution 141
VIII. The Vitality of the Peoples of America 177
IX. Trends of Vital Indices 253
X. Constitution and Tuberculosis 273
XI. The Influence of Physical Activity upon Mortality 298
XII. The Longevity of the Parents of the Tuberculous and the Cancerous 355
XIII. Comparing the Mortality of Man with that of Other Animals 372

PART III

Public Health and Epidemiology

XIV. National Food Consumption 383
XV. Food Waste 422
XVI. An Index of the Age Distribution of a Population 433
XVII. Some Biological Factors in the Epidemiology of Influenza 441
XVIII. The Incidence of Influenza among the Tuberculous 497
XIX. Epidemic Encephalitis 502
XX. The Statistical Evaluation of Public Health Activities 512

PART IV

The Population Problem

XXI. The Population Problem 523
XXII. Biology and War 534
XXIII. The Stability of England's Population Growth 550
XXIV. The Curve of Population Growth 558
XXV. The Growth of Human Populations 584
Index 639

LIST OF ILLUSTRATIONS

PAGE

FIG. 1. Variation in the brain-weight of Swedish males 46
2. Probable brain-weight for given age. Swedish ♂ total 61
3. Probable brain-weight for given age. Swedish ♀ total 61
4. Probable brain-weight for given age. Hessian ♂ total 61
5. Probable brain-weight for given age. Hessian ♀ total 62
6. Probable brain-weight for given age. Swedish ♂ young 62
7. Probable brain-weight for given age. Swedish ♀ young 62
8. Probable brain-weight for given age. Hessian ♂ young 63
9. Probable brain-weight for given age. Hessian ♀ young 63
10. Probable brain-weight for stature. Swedish ♂ total 67
11. Probable brain-weight for stature. Swedish ♀ total 67
12. Probable brain-weight for stature. Swedish ♂ young 67
13. Probable brain-weight for stature. Swedish ♀ young 67
14. Probable brain-weight for stature. Hessian ♂ total 68
15. Probable brain-weight for stature. Hessian ♀ total 68
16. Probable brain-weight for stature. Hessian ♂ young 68
17. Probable brain-weight for stature. Hessian ♀ young 69
18. Probable brain-weight for given body-weight. Bavarian ♂ total 74
19. Probable brain-weight for given body-weight. Bavarian ♀ total 74
20. Probable brain-weight for given skull-length. Bohemian ♂ young 77
21. Probable brain-weight for given skull-breadth. Bohemian ♂ young 78
22. Probable brain-weight for given skull-length. Bohemian ♀ young 79
23. Probable brain-weight for given skull-breadth. Bohemian ♀ young 79
24. Relative importance of the different organ systems of the body in human mortality 163
25. Percentages of biologically classifiable human mortality resulting from breakdown of organs developing from the different germ layers 169
26. Map showing states in the Birth Registration Area in 1919 185
27. Percentage of mixed effective marriages in 1919, and percentages of foreign born whites in 1910 194
28. Same as Fig. 27, but on native × native base 195
29. Relation between (a) proportion of foreign born whites in 1910, and (b) percentage which "fusion" effective matings are of effective all-foreign matings 197
30. Relation between (a) percentage of foreign born in 1910, and (b) proportion of foreign × foreign matings to native matings, effective in 1919 198
31. Proportion of children produced in the Birth Registration Area in 1919, from different types of matings 203
32. Relation between the still-birth rate and the total number of children which have been born 214
33. Specific death rates for males (by nativity) in six selected states in 1910 .. 218
34. Specific death rates for females (by nativity) in six selected states in 1910. 219

PAGE

35. Age specific vital indices for native and foreign born women in 1919..... 237
36. Age specific vital indices for males..... 240
37. Vital index A, in relation to age distribution of native born female population..... 242
38. Vital index B, in relation to age distribution of foreign born female population..... 243
39. The course of vital indices A and B from 1915 to 1921, inclusive, in the original birth registration states..... 261
40. The course of the vital indices A and B from 1915 to 1921, inclusive, in Maine..... 263
41. The course of the vital indices A and B from 1915 to 1921, inclusive, in New Hampshire..... 264
42. The course of the vital indices A and B from 1915 to 1921, inclusive, in Vermont..... 265
43. The course of the vital indices A and B from 1915 to 1921, inclusive, in Massachusetts..... 266
44. The course of the vital indices A and B from 1915 to 1921, inclusive, in Connecticut..... 267
45. The course of the vital indices C and D from 1915 to 1921 in southern birth registration states..... 269
46. The course of the vital indices C and D from 1915 to 1921, in the District of Columbia..... 271
47. Pedigree chart of a tuberculous family..... 274
48. Contact relations of tuberculous and non-tuberculous individuals of different grades of tuberculous ancestry..... 289
49. Course of the death rate from tuberculosis in the provinces of British Columbia, Saskatchewan, and Ontario, Canada..... 292
50. Frequency distribution of observed positional standard deviations for 95 chiefly indoor occupations..... 320
51. Like Fig. 50 but for 37 chiefly outdoor occupations..... 322
52. Differences in the mortality rates between heavy and light indoor occupations..... 333
53. Like Fig. 52, but for outdoor occupations..... 339
54. Age specific death rates of males engaged in light indoor and light outdoor occupations..... 340
55. Like Fig. 54, but for heavy occupations..... 341
56. Frequency polygons showing the age distribution of dead fathers of dead (a) tuberculous, and (b) non-tuberculous individuals..... 363
57. Like Fig. 56, but for living fathers of dead individuals..... 364
58. Like Fig. 56, but for dead fathers of living individuals..... 366
59. Like Fig. 56, but for living fathers of living individuals..... 366
60. Frequency polygons showing the age distribution of dead mothers of dead (a) tuberculous, and (b) non-tuberculous individuals..... 367
61. Like Fig. 60, but for living mothers of dead individuals..... 367
62. Like Fig. 60, but for dead mothers of living individuals..... 368
63. Like Fig. 60, but for living mothers of living individuals..... 368
64. Survivorship distributions for (a) the rotifer *Proales decipiens*, (b) man, and (c) *Drosophila*..... 378

PAGE

65. Relative curves for human food consumption ... 395
66. Percentages of the total nutritional intake of the American people derived from primary and secondary sources ... 397
67. The relative proportions of the American and British food intake derived from animal sources ... 398
68. Percentage contribution of the different great food commodity groups to the nutritional intake of the United States ... 399
69. Increase (or decrease) in food consumption in 1917–18 as compared with the average of the preceding six years ... 403
70. Percentage of the total protein consumed in the United States contributed by each of 23 commodities ... 408
71. Like Fig. 70, but for fat consumption ... 409
72. Like Fig. 70, but for carbohydrate consumption ... 410
73. Like Fig. 70, but for total energy value of food consumed ... 411
74. Percentage increase (or decrease) in consumption in 1917–18 as compared with the annual average of the six years preceding ... 414
75. Energy value in calories of the gross consumption of human food, per adult man per day ... 420
76. Seasonal curve of garbage production ... 427
77. Age distribution of population of Albany, N. Y. compared with stationary population ... 434
78. Like Fig. 77, but for Atlanta, Georgia ... 435
79. Spot diagram of correlation of age indices ... 439
80. Annual death rate, by weeks, per 1,000 population for eight cities ... 444
81. Like Fig. 80 ... 445
82. Like Fig. 80 ... 446
83. Like Fig. 80 ... 447
84. Like Fig. 80 ... 448
85. Like Fig. 80, but for 2 cities ... 449
86. Distribution of maximum influenza peak death rates in 40 cities ... 454
87. Distribution of peak dates of the epidemic ... 456
88. Frequency of occurrence of second and third peaks of mortality at different dates ... 459
89. Frequency of different durations of the first outbreak of epidemic mortality. 464
90. Cumulated frequency curves for time covered by (a) ascending limb, and (b) descending limb of epidemic mortality curve ... 466
91. Hypothetical diagram to show epidemic of great explosiveness ... 469
92. Hypothetical diagram to show epidemic of small explosiveness ... 469
93. Outline map of the United States ... 481
94. Regression line of 25-week excess mortality rate of influenza epidemic on explosiveness of outbreak of epidemic mortality ... 494
95. Progress of the world's population and production of certain materials in the nineteenth century ... 526
96. Progress in world food production in relation to population ... 530
97. Trend of vital index and crude birth rate in England and Wales, 1838–1920 inclusive ... 554
98. Observed and calsulat.on populations (from logarit.mic parabola) of the United States from 1790 to 1920 ... 564

PAGE

99. General form of curve of population growth 571
100. General form of equation (xiii) when n is even and a_n is positive 574
101. Like Fig. 100, but for n odd, and a_n negative 574
102. One form of skew growth curve 575
103. Growth curve of cycles, successive in time and additive 575
104. The population growth of the United States 591
105. The population growth of Austria 594
106. The population growth of Belgium 597
107. The population growth of Denmark 599
108. The population growth of England and Wales 601
109. The population growth of France 604
110. The population growth of Germany. Fitted with two symmetrical growth curves 608
111. The population growth of Germany. Fitted with the general curve for population growth 610
112. The population growth of Hungary 612
113. The population growth of Italy 614
114. The population growth of Norway 616
115. The population growth of Scotland 618
116. The population growth of Servia 620
117. The population growth of Sweden 622
118. The population growth of Japan. Fitted with single, symmetrical growth curve 624
119. The population growth of Japan. Fitted with general curve 626
120. The population growth of Java 628
121. The population growth of the Philippine Islands 630
122. The population growth of the world 633
123. The population growth of Baltimore (Old Area) 636

PART I

CONSIDERING MAN AS AN ANIMAL

CHAPTER I

The Weight of the Human Brain[1]

I. Introduction

The subject of brain-weight in man has for a long time been given considerable attention by anatomists and anthropologists. The reason for this is obvious. Since the brain is the organ of the mind it appeared to earlier workers that size of brain ought to be an index of intellectual capacity. The substance of the brain is of such physical homogeneity that its weight is a fairly accurate, as well as simple, "size" measure of the organ. Consequently, by earlier workers brain-weights were collected, studied and discussed to a considerable extent as measures or indices of brain power. It became evident after a time, however, that there was not the close and definite relationship between brain-weight and intellectual capacity which had been supposed to exist. Individuals of marked intellectual power were found in not a few instances to have brain-weights below the average, while on the other hand it was not at all difficult to find individuals of very mediocre intellectual attainment who possessed brains of unusually large size and weight. These results still held even after rough corrections were made for bodily size, age, etc. While it is thus evident that brain-weight cannot be taken as a close index of intellectual power holding for individual instances, yet it is clear that, considered from the phylogenetic standpoint, increase in brain-weight and in psychic capacity have in general gone hand in hand, and the weight of the brain increases quite regularly as we go up the taxonomic scale.[2]

The trend of investigation in this subject is rather from the point of view of anthropology than of psychology. Evidently brain-weight is

[1] This chapter first appeared under the title "Biometrical studies on man. I. Variation and correlation in brain-weight," in *Biometrika*, vol. 4, pp. 13–104, 1905. It is reprinted here without essential change, except that the extensive tables of raw data which formed an appendix to the original paper are not here reproduced. Any reader who may wish to consult them will find them *loco citato*, pp. 84–104.

[2] *Cf.* Ziehen, Th., "Das Gehirn, Massverhältnisse," Bardeleben's *Handbuch der Anatomie des Menschen*, Bd. iv, 1–3 Abtheilung, pp. 353–386, 1899, p. 362 *et seq.*; and Spitzka, E. A., "Brain-weight of animals with special reference to the weight of the brain in the Macaque monkey," *Jour. Comp. Neurol*, vol. xiii, pp. 9–17, 1903.

an important and interesting anthropological character, and in this field we may expect significant results.

The method of investigation which has been almost universally followed in brain-weight work has been to tabulate large masses of statistics of weighings, compute means for various groupings, and draw the conclusions which appeared to follow from the tabulations and averages. In other words the only statistical methods which in most cases have been applied to the data have been those of the sociological statistician. Such methods serve fairly well, of course, when only the "types" are wanted, but they are quite inadequate for some of the work which many neurologists have wished to do in this field. Practically all students of the subject have attempted to determine in one way or another the degree of correlation which exists between brain-weight and other physical characters and also age. Knowledge of these correlations is of course much to be desired. The human brain is justly to be regarded as the highest product of organic evolution. Any contribution to a knowledge of the laws governing its variation and correlation cannot fail to be of the greatest interest. Again, only through a knowledge of the degree of the correlation of brain-weight with other characters of the body is it possible to make scientifically such suitable corrections for bodily differences as will make fair any comparison of the brain-weights of different races, or of different groups of the same race. Now, as everyone knows who has even an elementary knowledge of statistics, it is possible to make the same statistical material lead to quite different conclusions, by grouping it in different ways, when the tabulations and averages are the only sources from which conclusions may be drawn. As a matter of fact this has happened in work on brain-weights. Different investigators, working in different ways, have arrived at quite different numerical appreciations of the relation existing between brain-weight and other characters. The very interesting question of variation in brain-weight had been scientifically investigated only by Pearson.[3]

The reason which led me to undertake the present piece of work was primarily a desire to apply adequate statistical methods to a biometrical problem of peculiar intrinsic interest, and for which fairly large collections of reliable data were available. It was desired to determine as a part of a general plan of work outlined as exactly as possible from the data at hand, what were the actual conditions of variation and correlation in

[3] Pearson, K., "Variation in man and woman," *The Chances of Death*, vol. i. pp. 319–323. Contemporaneously with the present work appeared a study of English brain-weights by Gladstone, R. J., "A study of the relation of the brain to the size of the head," *Biometrika*, vol. 4, pp. 105–123, 1905.

brain-weight. It was hoped that by such work some light might be thrown on the question of the evolution of man's brain. The work was originally planned to include the analysis of only one large collection of brain-weight data but when the constants for this material had been determined certain of the results were seen to be rather peculiar. Not being satisfied that these indicated the true state of affairs, but thinking that they might be caused by some hidden anomalies of the raw material, it was decided to extend the work to other data in order to test the first results.

The specific problems to which attention has been directed in this work are:

1. The amount of variation in the weight of adult male and female brains.
2. The relation of brain-weight to age in the adult.
3. The relation of brain-weight to stature and body-weight in the adult.
4. The relation of brain-weight to skull characters.
5. The nature of the regression of brain-weight on these other characters.
6. The effect of natural selection on brain-weight.
7. The value of brain-weight statistics from the anthropological standpoint.

II. DATA

The statistical data for this study were obtained from several sources, as has been mentioned above. The work was begun on Marchand's statistics, then extended to include the old, but still valuable collection of brain-weights by Bischoff. With the results from these two series in hand, I was still not satisfied that I had analyzed sufficient material to be certain of the conclusions. So I determined to include the following material in the discussion: (1) the admirable series of brain-weights of Swedes collected by Retzius. (2) Matiegka's Bohemian series. (3) The Boyd-Marshall data observed at the St. Marylebone Infirmary. As will appear later the last mentioned series was used only for a special purpose. The general results of this study are based on the analysis of the Marchand, Bischoff, Retzius and Matiegka series.

Marchand's[4] data were obtained from a series of brain-weighings made between the years 1885–1900 in the Pathological Institute at Marburg. The total number of brains weighed was 1234. Of this number 389 were from individuals under fifteen years of age, and hence were unavailable for adult brain-weight studies. The data recorded from each individual

[4] Marchand, F., "Ueber das Hirngewicht des Menschen," *Abhandl. d. math.-phys. Cl. d. Königl. Sachs. Gesellsch. d. Wiss.*, Bd. xxvii, No. iv, pp. 393–482, 1902.

in Marchand's work included, in addition to brain-weight, sex, age, and stature. It was not possible, however, to determine all of these points in every case, so that omissions of one or more records for an individual occur not infrequently in these statistics. The same is of course true of all extensive series of brain-weight data ever collected. After all such incomplete records have been thrown out there remain in Marchand's tables 475 male and 281 female complete records, or a total of 756 of both sexes. Marchand did not include body-weight in his determination, because he considered that: "das Körpergewicht bei dem Leichenmaterial der Krankenhäuser ein so wechselnder Faktor ist, dass bestimmte Beziehungen zum Gehirngewichte sich daraus kaum ableiten lassen würden."

The brains were weighed in the fresh condition, usually immediately after removal from the cranial cavity. The membranes were not removed before weighing. The weighings were made in the majority of cases to the nearest 5 grams, greater accuracy not being possible on account of such unavoidable sources of error as varying amounts of blood in the vessels of the brain, amount of fluid in the ventricles and membranes, etc. These sources of error cause variations in the total weight outside the limits of 5 grams. Marchand's material comes from the "hessische Bevölkerung."

Bischoff's[5] data comprise the results of the weighing of something over 800 brains of individuals between the ages of 17 and 85. The attempt was to determine for each individual the following characters in addition to brain-weight: sex, age, cause of death, stature and body-weight. Omissions in the records were fairly numerous, so it was only possible to extract full data on sex, stature, body-weight and brain-weight for 365 males and 241 females. This material I have referred to in this paper as the "short" Bavarian series. For determining the correlation between brain-weight and age I was able to extract 529 male and 323 female records from Bischoff's tables. This larger group I have called the "long" Bavarian series. The brains were weighed in the fresh condition, soon after removal and with the membranes. Nearly all the individuals had died in hospitals and a considerable number of them were convicts. Practically all were members of the middle and lower classes of society. The causes of death for this and Marchand's series were very varied, including a wide range of diseases and accidents, so that the material is not vitiated by the great preponderance of some one cause of death which might have a specific effect on brain-weight. The great majority of the individuals included in Bischoff's series were Bavarians.

[5] Bischoff, T. L. W. v., *Das Hirngewicht des Menschen*, Bonn, 1880, pp. vi. and 171, plus tables.

The Swedish data used were collected by Retzius.[6] The source of the material was in the main the autopsies at the great Sabbatsberg Krankenhaus in Stockholm, although some of the returns were from autopsies at the Maria Krankenhaus. This series of brain-weights is undoubtedly one of the best which has ever been made with reference to accuracy in the individual weighings and uniformity in the conditions and method of weighing. It is only to be regretted that the series is not more extensive. Regarding the racial homogeneity of the material Retzius says (*loc. cit.*, p. 55): "ich überzeugte mich aber in den allermeisten Fällen davon, dass sie eine echt schwedische Herkunft anzeigten." The method of weighing was as follows (*loc. cit.*, p. 55): "Die Gehirne wurden ohne Abnahme der weichen Hirnhaute, in der Regel kurz nach der Herausnahme, d.h. nur nach dem Verlust einer dabei stets ausfliessenden geringen Menge Blut und Cerebrospinalflüssigkeit, direct auf die Waageschale gelegt." The series as published by Retzius gives the weights of 450 male brains, and 250 female. In addition to brain-weight, age, stature and cause of death are recorded. After throwing out individuals incompletely recorded, and those falling outside the age limits 20 to 80, there was left available for this study 416 male records and 233 female.

Matiegka[7] has furnished some very valuable data for the student of brain-weight. His material came from the autopsies at two institutions in Prague, the Institut für gerichtliche Medizin, and the Pathologisch-Anatomische Institut. The series from the former source is the larger and more complete, and is the only one which is used in the present discussion. This includes the brain-weights of 372 males and 197 females, together with records of age, stature, skull-length and skull-breadth. The individuals were all adults between the ages of 20 and 80. The great majority of them were Czechs. The method of weighing the brain was that usually followed: the pia and arachnoid were not removed before weighing. The special value of this material of Matiegka's lies in the fact that it is the most extensive series of brain-weights in which skull characters are so recorded as to make it possible to determine their correlation with the weight of the brain. In addition, data are given for the determination of the correlation between brain-weight and stature, and brain-weight and age. Unfortunately Matiegka's material is open to criticism in certain

[6] Retzius, A., "Ueber das Hirngewicht der Schweden," *Biol. Untersuchungen*, N. F., Bd. ix., Cap. iv, pp. 51–68, 1900.

[7] Matiegka, H., "Über das Hirngewicht, die Schädelkapacität und die Kopfform, sowie deren Beziehungen zur psychischen Thätigkeit des Menschen," *Sitzber. des kön. böhmischen Gesellsch. d. Wissensch. Mathem.-Naturwiss. Classe*, Jahrg., 1902, No. xx, pp. 1–75.

important respects. In the first place detailed tables showing the exact records for each individual are not given, but instead the material is thrown into the form of correlation tables. In making up these tables the units of grouping were not altogether wisely chosen. This results in introducing a certain error into the absolute values of the constants deduced from these tables. The exact cause, nature and amount of this error will be pointed out later in the paper. Again, the skull-length and skull-breadth were measured in a very unreliable manner. Regarding the method of making these measurements Matiegka says (*loc. cit.*, p. 46): "Bei dem von mir verarbeiteten Materiale und zwar in beiden Instituten wurde gewöhnlich auch die *Länge* und *Breite* des *Schädeldaches* d. i. an dem behufs der Hirnentnahme vorgenommenen *Horizontalschnitte* gemessen. Das so erlangte Längenmass ist daher bedeutend *kürzer* als die *grösste Schädellänge*, die Breite wohl häufig etwas *kleiner* als die grösste Schädelbreite."[8] The effect of this procedure on the biometric constants is discussed later in this chapter. My chief reason for including this somewhat questionable material was for the sake of comparison, and because reasonably long series of brain weighings are not so plentiful as to allow one who would study the subject a great range of choice in material.

The data on the brain-weight of the English were very kindly placed at my disposal by Prof. H. H. Donaldson. He was able to obtain, some years ago, a copy of the original detailed manuscript tables which the English anatomist Marshall had compiled from Boyd's original data collected at the St. Marylebone Infirmary and the Somerset County Lunatic Asylum. These detailed tables were never published, though it was on them that Marshall's well-known paper, "On the Relations between the Weight of the Brain and its Parts and Stature and Mass of the Body in Man,"[9] was based. For the privilege of examining and using this unique and valuable material I am very grateful to Professor Donaldson.

From the biometrical standpoint the raw material available for a statistical study of the weight of the brain is peculiarly complex. The great bulk of the statistics consists of material gathered at the autopsies performed in large general hospitals or other public institutions of similar character. The result of this is that the only large collections of brain-weight data available are not representative "random" samples of the general population. Instead they represent a group of the population which has been subjected to a rather stringent selective process of a peculiar kind. It would be very difficult, if not impossible, to formulate all the

[8] Italics Matiegka's.

[9] *Jour. Anat. and Physiol.*, vol. xxvi, pp. 445–500, 1892.

factors which contribute to the differentiation of the "general hospital population" from the "general population" of the same locality. Of some of them, however, we may be fairly certain. In the first place, there are many classes (social) of the general population which will never be represented in any significant proportion in the general hospital population. Furthermore, the nature of the injury or disease from which an individual is suffering in many cases determines whether the individual shall be in a hospital. On this point, Greenwood,[10] in an able discussion of the matter, says: "Evidently the population of a general hospital will chiefly consist of: (i) persons acutely ill, (ii) those suffering from surgical injuries or diseases, (iii) sufferers from medical affections requiring special treatment. Chronic maladies of old age, such as bronchitis, indeed, any highly chronic disease, will be under-represented in comparison with the general death-rate. Similarly, the number of cases of valvular heart disease and rarer disorders, such as *diabetes mellitus* or *insular sclerosis* and other nervous lesions, will be above the general average."

In addition to the selection which occurs in the formation of the general hospital population there is a still farther weeding out when we come to deal with autopsy records, for the reason that not every individual dying in a hospital is subjected to a post-mortem examination. Whether there shall be an autopsy or not in a given case depends on several factors, one of which is the cause of death. The individuals whose last illnesses have given doubtful or rare clinical features are more likely to be subjected to a post-mortem examination, other things being equal, than the individuals in whose case the fatal illness has run a perfectly typical clearly cut course of some common disease. Consequently there is a tendency for the statistics to be unduly weighted with deaths from some of the rarer maladies. There can be no doubt that the statistical constants relating to any character of the body which is in any way affected by the disease causing death will be different, in material coming from hospital and pathological laboratory autopsy returns, from those which would be obtained could we get a perfectly random sample of the general population dying outside a hospital, and subjected to post-mortem examination regardless of the cause of death.

These peculiarities attendant upon hospital autopsy records are of particular importance when the character studied is the weight of the brain, for the reason that this character is definitely affected both by the

[10] Greenwood, M., "A first study of the weight, variability and correlation of the human viscera, with special reference to the healthy and diseased heart," *Biometrika*, iii, pp. 63–83, 1904, p. 65.

age of the individual, and, in certain cases, by the nature of the disease causing death. It is evident that there will be a more or less definite relation between the nature of the cause which brings an individual into a general hospital and the age of the individual. This combined relation of age and disease is a factor of importance in analyzing brain-weight data. In what manner will be apparent from a moment's consideration. Suppose we take the first of Greenwood's classes of the general hospital population mentioned above, namely, "persons acutely ill." It is at once clear that this will include two sub-groups. First in number and importance will be the group of individuals suffering from diseases medically classified as "acute" in the strict sense of the term, for example, typhoid fever. Second will be the group of individuals acutely ill at the termination of a long-standing chronic disease. These individuals in most instances have been cared for at home as long as the disease remained in the chronic or sub-acute form and only go into the hospital when it becomes acute. A good example of such cases is afforded by Bright's disease. There can be little doubt on *a priori* grounds that the mean age of the individuals will be lower in the first as compared with the second of these groups. This is shown to be the case by the actual statistics in the brain-weight series where "cause of death" is tabulated. In the lower age groups the causes of death returned are preponderantly "acute" in the strict sense. In the higher age groups we get a preponderance of the chronic affections. Now it is altogether likely that the acute illness which leads to death in a comparatively short time has much less effect on the weight of the brain than the chronic "wasting" sickness. So, then, the general result is that in the higher age classes where the brain-weight has decreased as the natural result of senescence there is a further artificial lowering on account of the preponderance of individuals who have been afflicted with "wasting" diseases. On the other hand the returns during early adult life give mean values for the brain-weight which are probably nearer the true normal value for the general population.

In the present study it was desired to consider only adult brain-weights and consequently only material which fell in age between 20 and 80 years was usually used. It is the general opinion of anatomists (*cf.* for example, Ziehen, *loc. cit.*, p. 359, and Marchand, *loc. cit.*, p. 404), that after the age period 15 to 20 there is very little increase of weight in the brain with advancing age. In other words the brain is considered to attain practically its complete growth in about the first 20 years of life. According to Marchand (*loc. cit.*, p. 402 *et seq.*) the maximum brain-weight remains practically constant till the individual is about 50 years old. After that age he be-

lieves that senile degeneration begins. Accordingly, in the discussion of the Marchand data, I have considered the period from 15 to 80 years to constitute the "adult period," so far as brain-weight is concerned. As a matter of fact, the result would have been but little different had the period 20 to 80 years been used in this case (as it was with all the other data), because there are only 36 males and 17 females falling in the age class 15 to 19, and these individuals are fairly evenly distributed among the brain-weight classes. When the work was begun I had, of course, no means of knowing how it would turn out, so it was necessary to decide how to treat the material on the basis simply of a careful general inspection of the statistics and of the opinion of other workers on the subject. As one point which I wished especially to investigate was the change of brain-weight with age, it became a nice problem as to how to handle the material so as to best get at this. I finally decided after a good deal of consideration to adopt provisionally Marchand's view stated above, viz., that the weight of the brain reaches a maximum between the ages 15 and 20, remains constant till about age 50, then declines through the old age period. Acting on this plan I separated the material in each case into what I have called a "young" and the "total" group. The "young" series included the individuals falling between the ages 20 to 50 (in the Hessian material, 15 to 50, and in the Bohemian 20 to 59 were the limits, in the former case from choice, in the latter from necessity). The "total" series included all the individuals between ages 20 and 80 (in the Hessian series 15 was the lower limit). The results show that on the whole this method of handling the material was adequate, considering the ends to be gained and the amount of material available.

III. FUNDAMENTAL CONSTANTS

As a preliminary to the discussion of the correlation of brain-weight with other characters it is necessary to exhibit and discuss the constants, measuring type and variability of the characters considered. Certain problems which are of considerable interest on their own account present themselves here. Of first importance perhaps is the question as to the amount of differentiation in respect of brain-weight which exists between different sub-races of men, both in type and variability. Further, the rather extensive material worked over makes it possible to settle the problem of the relative variability of the sexes with respect to the weight of the brain. On this point Pearson (*loc. cit*) has made a brief communication based on an analysis of four short series of English data, Bischoff's

Bavarian series, and a French series. His general conclusion is that in respect of brain-weight the sexes are sensibly equally variable. Another interesting problem which merits discussion here is as to how the relative variability in brain-weight compares with the variability of other organs and characters of the body.

In table 1 are exhibited the values of the Means, Standard Deviations and Coefficients of Variation together with their probable errors for each of the characters studied in this work. While, of course, the primary object of study is the weight of the brain, yet it is necessary for the correlation determinations that we have the fundamental constants for the other characters, age, stature, etc. To effect economy of space the constants for these other characters have been included in this first table. The frequency distributions from which the constants have been deduced will be found in the fundamental tables, numbered in Arabic numerals from 1 to 44, collected at the end of the original paper. Regarding the calculation of the constants the following explanations should be made. In preparing the correlation tables from the raw statistical material the following values for the units of grouping were chosen, and used uniformly throughout the work, except in certain cases where it was impossible to keep to them.

CHARACTER	STANDARD UNIT OF GROUPING
Brain-weight	50 grams
Age	5 years
Stature	3 centimetres
Skull length	5 millimetres
Skull breadth	5 millimetres
Body-weight	5 kilograms

These standard units were chosen after considerable study and experimenting and are believed to be the best values for the data discussed. However, it has been shown recently by other workers[11] that "the system of grouping adopted is within wide limits immaterial." An examination of those cases recorded in the present paper where it was necessary to use different units of grouping—notably in the Matiegka data where the brain-weights were grouped into 100 gram classes and the ages into 10 year classes—leads to the same conclusion.

[11] "Assortative mating in man." A cooperative study, *Biometrika*, vol. ii, pp. 481–498, 1903.

The standard deviation was evaluated according to the equation $\sigma = \sqrt{\mu_2}$. In obtaining μ_2 Sheppard's correction was used throughout for the characters brain-weight, stature, skull length, skull breadth and body-weight. The uncorrected "rough" moment was used in getting the standard deviation in age, as in this case there is no approach to high contact at either end of the range.

The decimals have been retained to three places in the tabulated values in order that the significant value of the first place may be seen. It will be understood, of course, that in the computations the decimals were retained to a larger number of places for arithmetical reasons.

IV. BRAIN-WEIGHT TYPES

In brain-weight work hitherto it has not been possible to make accurate comparisons of the mean weight of the brain in different races. There were two reasons for this: one that the probable errors of the means were not determined, and the other that no method was available by which allowance could be made for the differences in the mean stature and age of the samples of material to be compared. Obviously if brain-weight is correlated with stature and age, we should expect to get a considerable difference between the mean brain-weight of two groups which differed widely with respect to these other characters. Inasmuch as a knowledge of the degree of correlation between these characters affords a method whereby the material can be reduced to a "standard stature-age base," it will be worth while to examine the actual differences in brain-weight types in the four racial groups here discussed.

Taking the gross values given in table 1 the following table 2 has been prepared to facilitate comparison.

It is apparent at once that, considering the magnitude of the probable errors, there are significant differences between the Bavarians, Swedes, and Bohemians, with respect to brain-weight, in both sexes and age classes. Between the Swedish and Hessian means the differences are smaller, and as the following table shows, are not significant when their probable errors are taken into account:

Total males:	Swedish mean—Hessian mean =	8.744 ± 4.952
Total females:	Hessian mean—Swedish mean =	7.193 ± 6.065
Young males:	Swedish mean—Hessian mean =	9.511 ± 6.331
Young females:	Hessian mean—Swedish mean =	10.714 ± 8.197

In no case is the difference as great as even twice its probable error, hence we must conclude that the differences exhibited might, so far as the extent of material allows us to judge, be due to random sampling.

TABLE 1

Fundamental constants

RACE	CHARACTER	MEAN						STANDARD DEVIATION		COEFFICIENT OF VARIATION	
		No.	♂	Table	No.	♀	Table	♂	♀	♂	♀
Swedes	Brain-weight (Total)	416	1400.481±3.516	1	233	1252.682±4.452	2	106.329±2.486	100.757±3.148	7.592±0.179	8.043±0.253
Swedes	Brain-weight (Young)	262	1415.267±4 550	3	127	1269 488±6.317	4	109.180±3.217	105.543±4.467	7.714±0.229	8.314±0.354
Hessians	Brain-weight (Total)	475	1391.737±3 487	13	281	1259 875±4.119	14	112.675±2.466	102.368±2.642	8.096±0.178	8 125±0 233
Hessians	Brain-weight (Young)	291	1405.756±4.402	15	173	1280 202±5.223	16	111.328±3.113	101.841±3.693	7.919±0.223	7 955±0 290
Bohemians	Brain-weight (Total)	372	1454.839±3 973	27	197	1310.914±4.650	28	113.608±2.813	96.772±3.288	7.809±0.197	7.382±0.252
Bohemians	Brain-weight (Young)	266	1460.150±4.731	25	133	1313.910±5.453	26	114.395±3.345	93.227±3.855	7.835±0.231	7.095±0.295
Bohemians	Brain-weight (Skull)	299	1463.712±4.503	29	159	1321 698±5.073	30	115.440±3.159	94.840±3.587	7.887±0.219	7.176±0.273
Bavarians	Brain-weight (Total, Long)	529	1363.185±3.245	37	323	1220.356±3.820	38	110.614±2.295	101 776±2.701	8.118±0.122	8.340±0.223
Bavarians	Brain-weight (Young, Long)	365	1369.110±3.841	39	238	1235.504±3.997	40	108.776±2.716	91.421±2.826	7.945±0.200	7.399±0.230
Bavarians	Brain-weight (Total, Short)	365	1357.466±3.888	35	241	1218.776±4.616	36	120.128±2.749	106.246±3.264	8.849±0.223	8.717±0.270
Swedes	Stature (Total)	416	169.789±0 225	1	233	158.710±0.297	2	6.807±0.159	6.719±0.210	4.009±0.094	4.233±0.133
Swedes	Stature (Young)	262	170.176±0 280	3	127	159.673±0.418	4	6.728±0.198	6.987±0.296	3.954±0.117	4.375±0.186
Hessians	Stature (Total)	475	167.359±0.223	13	281	156.178±0.277	14	7.189±0.157	6.895±0.196	4.296±0.094	4.400±0.125
Hessians	Stature (Young)	291	167.294±0.282	15	173	156.980±0.384	16	7.118±0.199	7.490±0.272	4.255±0.119	4.771±0.173
Bohemians	Stature (Young)	266	169.417±0.303	25	133	157.650±0.418	26	7.324±0.214	7.152±0.296	4.323±0.127	4.537±0.188
Bavarians	Stature (Total, Short)	365	166.549±0.226	35	241	154.712±0.270	36	6.393±0.160	6.213±0.191	3.838±0.096	4.016±0.124
Swedes	Age (Total)	416	45.024±0.452	5	233	47.629±0.671	6	13.671±0.320	15.195±0.475		
Swedes	Age (Young)	262	36.317±0.327	7	127	35.689±0.480	8	7.847±0.231	8.027±0.340		
Hessians	Age (Total)	475	42.889±0.521	17	281	44.795±0.656	18	16.843±0.369	16.304±0 464		
Hessians	Age (Young)	291	31.572±0.390	19	173	34.061±0.497	20	9.861±0.276	9.685±0.351		
Bohemians	Age (Total)	372	45.699±0.523	27	197	41.447±0.792	28	14.957±0.370	16.482±0.560		
Bavarians	Age (Total, Long)	529	43.171±0.396	37	323	40.333±0.561	38	13.495±0.280	14.954±0.397		
Bavarians	Age (Young, Long)	365	35.582±0.255	39	238	32.731±0 340	40	7.216±0.180	7.768±0.240		
Bohemians	Skull length (Young)	299	176.547±0.287	29	159	170.142±0.294	30	7.368±0.203	5.500±0.208	4.173±0.115	3.233±0 122
Bohemians	Skull breadth (Young)	299	149.841±0.260	31	159	144.953±0.296	32	6.654±0.184	5.530±0.209	4.441±0.123	3.815±0.145
Bavarians	Body-weight (Total, Short)	365	49.925±0.376	41	241	42.894±0.461	42	10.646±0.266	10.601±0.326	21.324±0.608	24.715±0.804

Is this agreement between the Hessian and Swedish peoples in the matter of brain-weight real, or only apparent and the result of compensating differences in the other correlated characters? An answer to this question can be reached in two ways; either by reducing one set to the same "stature-age base" as the other and then comparing results, or by analyzing the effects on the brain-weight of the observed stature and age differences in the two sets of material. Both of these methods depend on the

TABLE 2

Brain-weight types

	HESSIANS	BAVARIANS	SWEDES	BOHEMIANS
Total males:				
Mean brain-weight	1391.737	1363.185	1400.481	1454.839
Mean age	42.889	43.171	45.024	45.699
Mean stature	167.359	166.549	169.789	
Young males:				
Mean brain-weight	1405.756	1369.110	1415.267	1460.150
Mean age	31.572	35.582	36.317	40.545*
Mean stature	167.294		170.176	169.417
Total females:				
Mean brain-weight	1259.875	1220.356	1252.682	1310.914
Mean age	44.795	40.333	47.639	41.447
Mean stature	156.688	154.712	158.710	
Young females:				
Mean brain-weight	1280.202	1235.504	1269.488	1313.910
Mean age	34.061	32.731	35.689	35.613*
Mean stature	156.980		159.673	157.650

* These values were calculated by taking the first four age classes of the "total" series. It includes more individuals than the "young" series for the other characters. This was the only way in which the mean age of the "young" series in this case could be even approximated to, as the raw material given by Matiegka is incomplete in this respect. The age as given is probably a little too high, but the error cannot be great.

use of characteristic equations, the derivation of which will be described further on. For the present I shall make practical use of such equations without further discussion of their derivation or validity. Table 2 shows that in the samples with which we are dealing the Hessians, both male and female, average *younger* and *shorter* than the Swedes. Reducing the Hessians to the same "stature-age base" as the Swedes, by means of the appropriate equations given on page 71 *infra*, the results shown in table 3 are obtained.

These differences after the stature-age corrections are made are extremely small. This is particularly well shown if they are reduced to a relative basis, by expressing the differences as percentages of the observed Swedish brain-weight:

The difference between Total Swedish ♂♂ and Hessians (calculated) = 0.29 per cent of the observed Swedish brain-weight.

The difference between Total Swedish ♀♀ and Hessians (calculated) = 0.44 per cent of the observed Swedish brain-weight.

The difference between Young Swedish ♂♂ and Hessians (calculated) = 0.55 per cent of the observed Swedish brain-weight.

The difference between Young Swedish ♀♀ and Hessians (calculated) = 1.11 per cent of the observed Swedish brain-weight.

TABLE 3

		EQUATION
The probable brain-weight of a group of Hessian ♂ ♂ having the same age and stature as the Swedish ♂ ♂ (Total)..	= 1396.480	(23)
Swedish brain-weight observed.....	= 1400.481	
Swedish *heavier.* Difference =	4.001	
The probable brain-weight of a group of Hessian ♀ ♀ having the same age and stature as the Swedish ♀ ♀ (Total).....	= 1258.232	(25)
Swedish brain-weight observed.....	= 1252.682	
Hessian *heavier.* Difference =	5.550	
The probable brain-weight of a group of Hessian ♂ ♂ having the same age and stature as the Young Swedish ♂ ♂.....	= 1407.457	(24)
Swedish brain-weight observed.....	= 1415.267	
Swedish *heavier* Difference =	7.810	
The probable brain-weight of a group of Hessian ♀ ♀ having the same age and stature as the Young Swedish ♀ ♀.....	= 1283.540	(26)
Swedish brain-weight observed.....	= 1269.488	
Hessian *heavier.* Difference =	14.052	

It appears then that, so far as the available material may be considered valid as representing the whole population, the conclusion is justified that the Hessian and Swedish peoples are sensibly alike in respect to their brain-weight. The agreement would be closer than that actually observed if the assumption made in the course of reasoning here followed, that the regression of brain-weight on age and stature is strictly linear, were exactly true. As will be shown later these regressions are not strictly linear but they approach linearity with sufficient closeness to serve for all

practical purposes. This, together with the difference in age distribution of the "total" and "young" series, also accounts for the fact that while in the "total" series and male "young" series the gross differences between Swedes and Hessians are lowered when we reduce to a common "stature-age base," these differences are slightly increased, on the other hand, when we deal in the same way with the shorter female "young" series.

This is in agreement with the general fact that these peoples are probably the most closely related ethnically of any with which we are dealing. The Swedes may be considered to be among the purest representatives of the original blonde, dolichocephalic Teutonic race (Ripley[12] and Deniker[13]). In the case of the Hessians some intermixture of this Teutonic with the brachycephalic, characteristically brunette Alpine type has occurred. The differentiation from the Swedes in such important characters as skull form and stature is not great however.[14]

Turning now to the other racial groups discussed, viz., the Bavarians and Bohemians (Czechs), we find as would be expected, that the differences in brain-weight are greater. Using the Hessian data as a basis for comparison, the gross differences with their probable errors are exhibited in the following table:

Total males:	Hessian mean—Bavarian mean = 28.552 ± 4.763
Total females:	Hessian mean—Bavarian mean = 39.519 ± 5.618
Young males:	Hessian mean—Bavarian mean = 36.646 ± 5.842
Young females:	Hessian mean—Bavarian mean = 44.698 ± 6.577
Total males:	Bohemian mean—Hessian mean = 53.102 ± 5.286
Total females:	Bohemian mean—Hessian mean = 51.039 ± 6.212
Young males:	Bohemian mean—Hessian mean = 54.394 ± 6.462
Young females:	Bohemian mean—Hessian mean = 33.708 ± 7.551

In all cases the differences are seen to be well above what might arise from errors in statistical sampling. Reducing to a common "stature-age base" by the method followed above the results in table 4 are obtained.

This table brings out several points of considerable interest. In the first instance is to be noted the general effect of reducing the Hessians to the same "stature-age base" as the other races, upon the interracial differences in mean brain-weight. In the case of the Bavarians the deviations from the Hessian means are reduced slightly in both male series,

[12] Ripley, W. Z., *The Races of Europe*, New York, 1899, pp. xxxii, 624.

[13] Deniker, *The Races of Man*, London and New York, 1900, pp. xxiii, 328 and 611.

[14] This is well shown graphically in the maps indicating the distribution of stature and cephalic index in Europe, given by Ripley (*loc. cit.*, pp. 96 and 53). *Cf.* also Deniker *loc. cit.*, pp. 328 and 329.

and the "young" female series, when stature and age differences are eliminated. In the "total" female series the difference is increased over the original gross difference. The explanation for this discrepancy in the female "total" series is to be found in a peculiar abnormality which this series shows in its elemental frequency distribution, and which will be discussed later (p. 45). In the case of the Bohemians we get the somewhat remarkable result that a reduction to a common "stature-age base" actually increases the differences of this racial group in brain-weight as compared with the Hessians. Or in other words, in the samples with which

TABLE 4

		PROBABLE BRAIN-WEIGHT OF A GROUP OF BESSIANS OF THE SAME SEX, AGE AND STATURE AS THE	MEAN BRAIN-WEIGHT OBSERVED (BAVARIAN OR BOHEMIAN)	DIFFERENCE EXPRESSED AS IN EXCESS OR DEFECT OF HESSIAN VALUES	DIFFERENCE AS PER CENT OF OBSERVED MEAN (BAVARIAN OR BOHEMIAN)	EQUATION ON WHICH HESSIAN ESTIMATE IS BASED
					per cent	
Bavarian	♂♂, Total.....	1388.984	1363.185	−25.799	−1.9	No. 23, p. 71
	♀♀, Total.....	1265.137	1220.356	−44.781	−3.7	No. 25, p. 71
	♂♂, Young....	1397.157*	1369.110	−28.047	−2.0	No. 24, p. 71
	♀♀, Young....	1277.317*	1235.504	−41.813	−3.4	No. 26, p. 71
Bohemian	♂♂, Total.....	1394.570†	1454.839	+60.269	+4.1	No. 23, p. 71
	♀♀, Total.....	1259.310†	1310.914	+51.604	+3.9	No. 25, p. 71
	♂♂, Young...	1399.527‡	1460.150	+60.623	+4.2	No. 24, p. 71
	♀♀, Young...	1278.935‡	1313.910	+34.975	+2.7	No. 26, p. 71

* This assumes that the mean stature of the Bavarians would be the same in the "young" group as it is in the "total" group. The error introduced by this procedure is practically negligible.

† Here again, on account of lack of data, the mean stature is assumed to be the same in "total" and "young" groups.

‡ See footnote on p. 27.

we are dealing the stature and age differences act in a compensatory way and bring the mean brain-weights closer together than they would be if we dealt with selected samples of the populations, each sample having the same mean age and stature. The fact already noted (p. 20) that the mean brain-weights deduced from Matiegka's tables for the Bohemians are somewhat too large, may now be examined in detail. This Bohemian material was published by Matiegka in the form of correlation tables with unduly large units of grouping. The base unit for brain-weight was 100 grams. Now in calculating the general population mean

from these tables one assumes that the individuals in each elemental frequency group centre in brain-weight at the mid-point of that group. Thus the individuals recorded as having a brain-weight of between 1300 and 1400 grams are assumed to center at 1350 grams. But evidently this assumption will not be true except at the middle of the whole range. For example, the brain-weights recorded as between 1500 and 1600 will center somewhat below 1550. Now, of course, if the distribution is exactly or approximately normal and the number of individuals is sufficiently large, the errors from this cause on one side of the general population mean will balance those on the other, and we shall still be able to get a very close value for this mean from the frequency distribution. Unfortunately, in Matiegka's series, however, the total numbers are not sufficiently large to overcome entirely this error. So we have the following differences: by direct calculation from the individual observations Matiegka finds for the mean brain-weight of the "young" (20 to 59) series in the male 1450.4 grams, in the female 1305.5 grams. Our corresponding values are 1460.150 and 1313.910 grams or there is an excess of 9.7 and 8.4 grams respectively. For the "total" series (20 to 80) I have calculated from regression values which Matiegka gives (p. 7), based on the individual observations, general population means which give values as follows: males 1441.4 grams, females 1294.8 grams. The corresponding values from the tables are: males 1454.8 grams, females 1310.9 grams, or the excess of the table means is 13.4 grams for the males and 16.1 for the females. These differences are small and for practical purposes negligible.

The general conclusion may be drawn that, *apart from all differences in stature, and in the mean age of the samples studied*, the Bavarian mean brain-weight is lower than the Hessian (roughly about 2 per cent), and the Bohemian mean brain-weight is higher than the Hessian (roughly about 3.5 per cent).

These results seem to be of some importance as indicating a method whereby brain-weight may have some scientific validity as an anthropological character. This can hardly be said to be the case when nothing more is done than to tabulate means without probable errors, and no attempt is made to get rid of disturbing stature and age effects. Interracial differences in brain-weight may mean something or nothing. With modern biometrical methods it is possible to measure exactly these differences, and when such methods are used it is the belief of the writer that brain-weight can become a really significant anthropological character. In the cases discussed here we see ethnic affinities and differences clearly reflected in the brain-weight. Of the four racial groups studied the two

most closely related in origin and general anthropological characters, the Swedes and the Hessians, have mean brain-weights sensibly identical. The Bavarians and Bohemians, though close neighbours geographically, arise from totally distinct ethnic stocks ("Alpine" and Slav respectively) and associated with this we find a wide difference in the weight of the brain.

On the question of the homogeneity of brain-weight statistics, some evidence is furnished in the third and fourth columns of table 4. If the inclusion of aged persons (50 years and over) introduced any considerable element of heterogeneity so far as brain-weight constants are concerned it would be expected that the interracial differences in the means would not be equal in the "total" and in the "young" series. As a matter of fact in the material here discussed they are sensibly equal, or perhaps better, they are not significantly different. The whole question of the homogeneity of this material will be treated fully in connection with the discussion of the variability in brain-weight.

The interesting questions brought up by this table of means, regarding sexual differences, association of brain-weight with other characters, etc., will be discussed in later sections.

V. VARIATION IN BRAIN-WEIGHT. HOMOGENEITY OF MATERIAL

Variability in the weight of the brain has been subjected to exact investigation by only one worker, Pearson (*loc. cit.*), so far as is known to the writer. He deduced from his material (cf. p. 23 *supra*) coefficients of variation ranging in value from 7.93 to 10.64 per cent, the higher values being from admittedly heterogeneous series.

The numerical values for the variation constants and their probable errors found in the present work are given in table 1.

The question of homogeneity of material should be first discussed. For reasons which have been set forth above it would be hopeless to look for any high degree of homogeneity in any collection of human brain-weighings at present available. The best we can hope for is a fair degree of homogeneity, and reasonably the same degree in different series which are to be compared. Unfortunately there is not available here, as in the case of craniometrical investigations, series in which fair homogeneity can be inferred with high probability, so that it is not possible to make a direct estimate by comparing variabilities with such a "known base." Instead resort must be had to indirect methods. The best of such indirect methods is based on the fact that if a random sample be taken from a *homogeneous* collection of material the variation constants for the sample and the whole

collection will not significantly differ. On the contrary if the material is non-homogeneous such a sampling will give different values for the constants. If the sample be *selected*, *i.e.*, not random, the variation constants for the character selected will of course be lowered. Now in the material as treated here we have in the "young" series for each racial group a *selection* from the "total" series, but a selection based on age, not on brain-weight directly. If there were no correlation between brain-weight and age such a selection on an age basis would be, of course, a random sample so far as brain-weight is concerned. Unfortunately for the argument, there is, as will be shown later, a sensible though in general low correlation between age and brain-weight. This being the case it would be expected that such a selection as has been made in separating out the "young" from the "total" series would result in a lowering of the

TABLE 5

Difference in variation constants between "young" and "total" series

	♂		♀	
	S. D.	C. of V.	S. D.	C. of V
Swedes	+2.851±4.066	+0.122±0.291	+ 4.786±5.465	+0.271±0.435
Hessians	−1.347±3.971	−0.177±0.285	− 0.527±4.541	−0.170±0.372
Bavarians	−1.888±3.556	−0.173±0.234	−10.357±3.909	−0.941±0.320
Bohemians	+0.787±4.371	+0.026±0.304	− 3.545±5.067	−0.287±0.388
Bohemians (Skull)*	+1.832±4.230	+0.078±0.295	− 1.932±4.866	−0.206±0.372

* See p. 19 and table 1 for the series here referred to.
Difference + when "Young" is greater.
Difference − when "Young" is less.

standard deviation and coefficient of variation in brain-weight. Now, as a matter of fact, as the following table shows, the correlation between brain-weight and age is so low that in the relatively small series under discussion, the "young" series forms practically a random sample of the "total" series, within the limits of error, with respect to brain-weight.

In only one case—the Bavarian females—is the difference as large as its probable error, and in this case the difference is not three times the probable error. The conclusion seems justified that the series are reasonably homogeneous in other respects than age. The age distribution is such as to exclude growth effects, and, as the differences indicate, the effects of senescence on brain-weight are so insignificant that for practical purposes the material may be considered sufficiently homogeneous to warrant further biometrical study. From the character of the differences it would

appear that the most homogeneous series are, on the whole, the Swedish and the Bohemian.

A direct examination of the standard deviations and coefficients of variation confirms the conclusion. In table 6 I have arranged in order according to ascending value, the variation constants for the "total" series, using as the basis for the grouping the coefficient of variation.

All of the four series used in this paper are in good agreement, both among themselves and with the skull capacity values. They are distinctly lower than the figures from the admittedly heterogeneous, Reid, Peacock, Sims and Clendinning English series, worked over by Pearson. Taking all the evidence together we are driven to the conclusion that the series are fairly homogeneous. If any one of our series is markedly hetero-

TABLE 6

Comparison of different races in respect to variation in brain-weight and skull capacity

	MALES			FEMALES	
	S. D.	C. of V.		S. D.	C. of V.
Swedes	106.329	7.592	Bohemians	96.772	7.382
Bohemians	113.608	7.809	Swedes	100.757	8.043
Hessians	112.675	8.096	Hessians	102.368	8.125
Bavarians	110.664	8.118	Bavarians	101.776	8.340
English*	124.48	9.20	English*	118.7	9.72
Skull Capacity, English†		8.28	Skull Capacity, English†		8.68
Skull Capacity, Germans‡		7.74	Skull Capacity, Germans‡		8.19

* Pearson: *The Chances of Death*, vol. i, p. 321.

† W. R. Macdonell: *Biometrika*, vol. iii, p. 221.

‡ Pearson: *The Chances of Death*, vol. i, p. 333.

geneous all the others must be equally so, which would be a very improbable result. That the series cannot be very heterogeneous in their make-up is also shown by the values of the coefficients of variation for skull capacity from two admittedly homogeneous series, the Whitechapel skulls studied by Macdonell, and Ranke's Bavarian series.

A comparison of the variability in brain-weight with that shown by other organs and characters of the human body may next be undertaken. I have arranged in the following table (7) the coefficients of variation for a variety of characters which have been studied by biometrical workers. The arrangement is in general that of descending order of values in the male series. The attempt has been made in the table to include representatives of all the different classes of organs and characters for which we have biometric data available.

The most noticeable and remarkable fact brought out by table 7 is that with the exception of capacity, all skull characters are roughly only about half as variable as brain-weight. Some such a relation as this might have been predicted, on the general ground that brain-weight measurements and statistics are on many accounts rather "loose," and would *indicate* a higher variability than would exact measurements on skulls, even though it had no real existence. Such reasoning, however, takes no account of the agreement, which is really remarkably close when we recall the numerous sources of error in brain-weight returns, between skull capacity and brain-weight in their variability. All will admit that capacity is the most difficult skull character to measure accurately, yet no one would maintain that the difference in variability between cephalic index, for example, and skull capacity was entirely, or even in any considerable part, due to the element of error in the measurement of the latter. The agreement in variability between skull capacity and brain-weight is, of course, to be expected on theoretical grounds. That it should turn out in practice to be so close is a first-rate guarantee of the general trustworthiness of brain-weight statistics.

As to the explanation of the great variability in brain-weight and skull capacity as compared with the other skull characters we may tentatively reason about the matter in the following way. If the list of organs and characters given in the foregoing table be examined a natural division into three groups almost immediately suggests itself. First, we have at the bottom of the list the "bone" measurements, in general including all those characters which depend primarily for their values on the dimensions of various parts of the skeleton. These characters give values for the coefficient of variation up to from 5 to 7, certain of the mandibular variabilities exceeding this limit. Next comes the group giving values for the coefficients of from 7 to 10, with the limits fairly sharply marked off. Finally we have all the characters giving values above 10. Now as one passes from one end of the series to the other a definite biological relationship is plain. At the upper end of the series (coefficient of variation 10 and over) the organs and characters tabulated are such as depend in a very considerable degree for their values as determined by measurement, on the general *metabolic condition of the organism* as a whole at the time the measurements are made or immediately before. This will be freely admitted, I think, for such characters as "Dermal sensitivity," "Keenness of sight," "Weight of body," "Swiftness of blow," "Strength of pull," "Breathing capacity" and the like. The same thing, though less apparent perhaps, is, I believe, true when the characters are the weights of viscera. Indeed

TABLE 7

Coefficients of variation for man

	♂	♀
Weight of Spleen (General Hospital Population)*	50.58	
Weight of Spleen (Healthy)†	38.21	
Dermal Sensitivity‡	35.70	45.70
Weight of Heart (General Hospital Population)*	32.39	
Keenness of Sight‡	28.68	32.21
Weight of Kidneys (General Hospital Population)*	24.63	
Weight of Body (Bavarians)	21.32	24.715
Weight of Liver (General Hospital Population)*	21.12	
Swiftness of Blow‡	19.4	17.1
Weight of Heart (Healthy)†	17.71	
Weight of Kidneys (Healthy)†	16.80	
Breathing Capacity‡	16.6	20.4
Strength of Pull‡	15.0	19.3
Weight of Liver (Healthy)†	14.80	
Height of Mandible (English, both sexes)§	11.73	11.73
Weight of Body (English)‡	10.37	13.37
Skull Capacity (Etruscan)‖	9.58	8.54
Brain-weight (French)‡	9.16	9.14
Skull Capacity (Modern Italian)‖	8.34	8.99
Skull Capacity (English)¶	8.28	8.68
Skull Capacity (Egyptian Mummies)‖	8.13	8.29
Brain-weight (Bavarian)	8.118	8.340
Brain-weight (Hessian)	8.096	8.125
Brain-weight (Bohemian)	7.809	7.382
Skull Capacity (Modern German)‖	7.74	8.19
Skull Capacity (Naqada)‖	7.72	6.92
Brain-weight (Swedish)	7.592	8.043
Skull Capacity (Parisian French)‖	7.36	7.10
Skull Capacity (Aino)‖	7.07	6.90
Mandible, Distance between Foramina mentalia (English, both sexes)§	6.23	6.23
Length of Forearm**	5.24	5.21
Length of Femur (French)‡	5.05	5.04
Length of Tibia (French)‡	4.975	5.365
Length of Humerus (French)‡	4.89	5.61
Length of Radius (French)‡	4.87	5.23
Skull, Height to Breadth Index (English)¶	4.86	4.16
Skull, Breadth to Height Index(English)¶	4.83	4.17
Length of Finger (English Criminals)††	4.74	
Skull, Ratio of Height to Horizontal Length (English)¶	4.61	4.10
Length of Foot (English)††	4.59	
Skull, Cephalic Index for Horizontal Length (English)¶	4.38	3.99
Length of Cubit (English Criminals)††	4.36	
Skull, Least Breadth of Forehead (English)¶	4.29	4.55

TABLE 7—*Continued*

	♂	♀
Skull, Height (English)¶	4.21	3.96
Skull, Length of Base (English)¶	4.07	4.11
Skull, Cephalic Index for Greatest Length (English)¶	3.95	4.03
Stature, (English)**	3.99	3.83
Skull, Ratio of Height to Greatest Length (English)¶	3.80	4.21
Skull, Greatest Breadth (English)¶	3.75	3.54
Skull, Auricular Height (English)¶	3.73	4.12
Skull, Face Breadth (English Criminals)††	3.707	
Skull, Cross Circumference (English)¶	3.70	3.97
Skull, Sagittal Circumference (English)¶	3.63	3.90
Head Breadth (English Criminals)††	3.333	
Skull, Length (English)¶	3.31	3.45
Head Length (English Criminals)††	3.154	
Skull, Horizontal Circumference (English)¶	2.87	2.92

* Greenwood, M., *Biometrika*, vol. iii, p. 66.
† *Ibid.*, p. 67.
‡ Pearson, K., *The Chances of Death*, vol. i, pp. 293–377.
§ Macdonell, W. R., *Biometrika*, vol. iii, p. 225.
‖ *Ibid.*, p. 221 (after Pearson).
¶ *Ibid.*, p. 222.
** Pearson and Lee, *Biometrika*, vol. ii, p. 370.
†† Macdonell, W. R., *Biometrika*, vol. i, p. 202.

Greenwood's own results show this to be the case, when we get such different results for means, variabilities and correlation according as we deal with the "general hospital population," "healthy" organs, or the same viscera in diseased conditions of different characters. Wynn[15] has shown that in rabbits there is a considerable degree of probability that continued administration of digitalis will in a short time raise the mean weight of the heart appreciably, while at the same time the body weight is lowered. It is to be regretted that his series of experiments included so (statistically) few individuals.

Furthermore, it seems reasonable to assume that in this upper group the thing measured in the majority, if not in all cases, is not the thing natural selection has acted upon directly, allowing that it has acted at all. I think it may be fairly assumed that so far as natural selection has acted at all on these organs and characters, the selection has been in the direction of *ability to function properly* so as best to conserve the physiological economy of the organism as a whole, rather than in the direction of abso-

[15] *Jour. Amer. Med. Assoc.*, vol. xliii, pp. 164, 165. 1904.

lute size of organ or character. The physiologically balanced functioning with reference to the needs of the organism as a whole is the important thing in such organs as the spleen, pancreas, liver, etc. Absolute size of organ can hardly be a very close direct measure of ability to function well. So then, I am inclined to attribute the high variability observed in those organs and characters falling together in the uppermost part of the table to these two factors: (*a*) the value obtained by measurement depends to a considerable degree on the general metabolic condition of the individual at the time, and (*b*) the thing measured is not the thing with which natural selection, so far as it has acted at all, has had directly to do.

On the other hand, if we turn to the lowest group in the above table (coefficients of variation under 5 to 7 for the males) we find the two factors just mentioned almost exactly reversed. Here the characters are either of the skeleton directly, or in the case of the anthropometrical data are characters which closely depend for their measured values on the size of portions of the skeleton. Obviously the general metabolic condition of the organism has, within wide limits, little effect on the length of the femur or the skull, for example, and if there is an effect it manifests itself very slowly. Further it is to be presumed that the absolute size is of greater direct selective value in the case of parts of the skeleton than in the case of organs like the liver, etc. In other words, size is much more directly related to proper functioning in the former than in the latter case. The skull may at first sight appear to form an exception here. It must be kept in mind, however, that the skull serves two very important—perhaps equally important—functions; viz. (1) the enclosing and protecting of the brain, and (2) the serving as a basis for the attachment of the complexes of muscles which actuate the masticatory apparatus and support and move the head. With reference to each of these factors natural selection may act on the skull. So far as the second factor at least is concerned, size of skull (in a broad sense) will be of selective value. So that here again, since size and function are interrelated, we should expect to find the results of selective action reflected in the size measurements of characters. In general, I think it can safely be maintained that the low variability shown in the group of characters under discussion is what might be expected to result from the operation of the factors just mentioned.

In the middle group (coefficients of variation ranging from 7 to 10 in the male) including brain-weight and skull capacity we seem to have also an intermediate condition, with respect to the two factors which have been mentioned as among the causes which contribute to the observed variability. In the first place, there can be no doubt in face of the evi-

dence both from the biometrical and anatomical standpoints, that the cranial capacity is quite highly correlated with volume of the brain.[16] Admitting this, the discussion may be restricted to the brain. Now it has been known from the time of the earliest collection of brain-weight statistics that the weight of the brain is influenced to a certain extent by the general metabolic condition of the individual preceding death. I am inclined to think however that the extent of this influence has been over-estimated. My primary reason for this view comes from the analysis of the statistics themselves. If the metabolic condition of the individual preceding death influenced brain-weight to a marked degree, one would hardly expect to find the agreement shown in the constants of variation and correlation tabulated here, when different groups of individuals with different distribution of ante-mortem affections were compared. We might expect by chance to get two groups in agreement, but on this supposition the odds would be great against getting four groups to agree so closely as they actually do. Furthermore, there is a certain amount of evidence from other sources indicating that the influence of ante-mortem general metabolic conditions on brain-weight, while always present as one factor, does not produce so marked an effect on the weight of the brain as on the size measurements of some of the organs in our most variable group. For example, Mühlmann[17] studied microscopically the brains of 24 individuals of different ages from birth to 90 years to determine the amount of pigment degeneration in the ganglion cells at the different ages. In the adults the causes of death included such divergent conditions as endocarditis, pneumonia cruposa, and nephritis chronica. Yet the differences found in the appearance of the brain in different cases were clearly associated with differences in age, and not with causes of death. On this whole question more data are needed. Exact measurements of the correlation between skull capacity and brain-weight are much to be desired. We also lack definite biometrical evidence regarding the effect of morbid conditions on the weight of the brain. The evidence at present available seems to lead to the conclusion here indicated.

On the other hand, it can hardly be maintained that natural selection has acted on the size of the brain to the degree that it has on skeletal structures. The brain, in this respect, comes nearer to the organs included in our most variable group. So that, on the one hand with a less marked dependence of the measurement on the previous metabolic condition of the organism than is found in the most variable group of organs and characters, and on the other hand, with a less stringent selection with reference

[16] On the anatomical side, *cf.* Symington, *Nature*, vol. 68, pp. 539–544.

[17] *Verhandl. d. deutsch. Pathol. Gesellsch.* Bd. iii, pp. 148–157.

to size as compared with the least variable group, an intermediate condition in variability for the brain is to be expected.

Of course, too much stress cannot be laid on this argument as to the relative variability of the characters of the human body in view of the meagreness of the data at present available. The data at hand, however, plainly suggest some such an explanation, and it is only as a tentative suggestion worthy of being kept in mind as further data are available that the matter has been developed here. To summarise; when a series of the various organs and characters of the body whose variation has been measured are arranged in the order of their relative variability, those characters falling at the most variable end of the series are those whose measured value is affected markedly by the previous general metabolic condition of the organism, and in which ability to function properly is not closely related to size of organ, and in which natural selection, if it has acted at all on the character, has not acted directly upon the thing measured. The opposite end of the series includes the least variable organs and characters, in which both the above mentioned conditions are reversed. Brain-weight and skull capacity occupy an intermediate condition both with reference to variability and the conditioning factors discussed.

Attention may be turned next to the question of the relative variability of the different races in respect to brain-weight. In the following scheme the races are arranged in descending order of mean brain-weight in the left-hand columns, and of variability (measured by the coefficient of variation) in the right-hand column. The lists are based on "total" series.

From these lists it would appear that as a general rule the higher the mean brain-weight is, the lower will be the variability. But it must be kept in mind that with our present series none of the differences in the variability columns is significant. Taking the extremes, Bavarians and Swedes for the males, and Bavarians and Bohemians for the females, the differences between the coefficients of variability with their probable errors are respectively 0.526 ± 0.217 and 0.958 ± 0.337. In these cases the difference is less than three times its probable error and cannot be considered as certainly significant. The lists are introduced merely

♂		♀	
Mean	C. of V.	Mean	C. of V.
Bohemians	Bavarians	Bohemians	Bavarians
Swedes	Hessians	Hessians	Hessians
Hessians	Bohemians	Swedes	Swedes
Bavarians	Swedes	Bavarians	Bohemians

to show the general trend of the results. It is possible that with much larger series of brain-weight statistics and consequently reduced probable errors some such relation as that just stated might be definitely proven.

For the sake of comparison and reference I include here tabular arrangements of the means and variabilities for the characters stature and age, made in the same way as that for brain-weight.

Stature

♂		♀	
Mean	C. of V	Mean	C. of V
Swedes	Bohemians	Swedes	Bohemians
Bohemians	Hessians	Bohemians	Hessians
Hessians	Swedes	Hessians	Swedes
Bavarians	Bavarians	Bavarians	Bavarians

Age

♂		♀	
Mean	S. D.	Mean	S. D
Bohemians	Hessians	Swedes	Bohemians
Swedes	Bohemians	Hessians	Hessians
Bavarians	Swedes	Bavarians	Swedes
Hessians	Bavarians	Bohemians	Bavarians

Comparing the different characters the means seem to be entirely chaotic, but there is more regularity as regards the variabilities. The relative variabilities in stature and age for the different samples run parallel, with the exception of the Hessians and Bohemians in the male series. The samples which show the greatest variability in age of individuals included, also show the greatest variability in stature, and *vice versa*. This relation is, of course, to be expected, but the relation between the brain-weight series and the other two is rather curious. There is no parallelism between the variability columns of the brain-weight series and the other two, but the variability columns of the stature series together with the females of the age series exactly agree (with the exception of the transposition of the Hessians and Swedes in the male brain-weight series) with the columns of *means* of brain-weight. In other words in those series showing the greatest variability in stature (and in the females of age also) we have the highest mean brain-weight, and *vice versa*. No reason for this curious parallelism is apparent, and it may be purely accidental, but it seems worth noting.

The questions of the relative variability of the sexes in brain-weight and the racial differences as affected by sex will be discussed in a later section.

VI. ON THE NATURE OF THE FREQUENCY DISTRIBUTION

In this section the variability in brain-weight will be discussed analytically, according to the methods and nomenclature of Pearson's fundamental memoir on Skew Variation[18] and its supplement.[19] My purpose in considering these curves analytically is not primarily that data may be furnished so that the material may be fitted with appropriate curves, but rather in order that definite knowledge may be had as to whether the variation in this character obeys the "normal" law of the deviation of errors. Both Miss Fawcett[20] and Macdonell[21] have reached the conclusion that, for practical purposes at least, the majority of skull characters may be considered to conform to this law in their variation. It is of prime importance to determine in how far the same is true of the weight of the brain. Since this was my chief object in analyzing the data, I shall not at this time deal graphically with the curves, but instead shall merely present the chief analytical constants arranged in tabular form. Furthermore, from considerations of the time involved in computation, I have not determined the analytical constants for all the brain-weight frequency distributions given. Instead, after examining all the data, I decided to confine myself to the "total" series for both sexes of the four races.

The analytical constants for the selected series are exhibited in table 8. In the second column is given the number of brains on which the calculation in each case is based. The third column gives the unit in terms of which the second, third and fourth moments about the mean (μ_2, μ_3, and μ_4) are calculated: the next two columns give β_1 and $\sqrt{\beta_1}$, and the two following, β_2 and $3-\beta_2$. Following this are given in order the "criterion" ($\kappa_1 = 2\beta_2 - 3\beta_1 - 6$),[22] the mean, mode and skewness. The skewness was calculated from the moments directly by the formula

$$Sk = \tfrac{1}{2}\frac{\sqrt{\beta_1}\,(\beta_2 + 3)}{5\beta_2 - 6\beta_1 - 9},$$ *

* Pearson, K.: "On the mathematical theory of errors of judgment and on the personal equation," *Phil. Trans.*, Vol. 198, A, pp. 235–299. Page 277.

[18] *Phil. Trans.* Vol. 186, A, pp. 343–414.
[19] *Ibid.* Vol. 197, A, pp. 443–459.
[20] *Biometrika*, Vol. i. p. 443.
[21] *Ibid.* Vol. iii. p. 227.
[22] *Phil. Trans.* Vol. 197, A, p. 444.

TABLE 8

Analytical constants of curves

Brain-weight

RACE AND SERIES		NO.	UNIT	μ_2	μ_3	μ_4	β_1	$\sqrt{\beta_1}$	β_2	$3-\beta_2$	CRITERION	MEAN	MODE	SKEWNESS
			grams											
Swede "Total"	♂	416	50	4.5223	1.6292	57.18995	0.0287	0.1694	2.7964	0.2036	−0.4934	1400.481	1389.627	0.1021
	♀	233	50	4.0608	1.8441	51.1703	0.0508	0.2254	3.1031	−0.1031	0.0539	1252.682	1241.526	0.1107
Hessian "Total"	♂	475	50	5.0783	−4.1458	86.8638	0.1312	0.3623	3.3682	−0.3682	0.3428	1391.737	1373.311	0.1635
	♀	281	50	4.1917	−0.2601	49.8514	0.0092	0.0958	2.8372	0.1628	−0.3531	1259.875	1254.294	0.0545
Bohemian "Total"	♂	372	100	1.2907	0.5908	5.2300	0.1623	0.4029	3.1396	−0.1396	−0.2078	1454.839	1430.292	0.2161
	♀	197	100	0.9365	0.1033	2.5144	0.01298	0.1139	2.8671	0.1329	−0.3048	1310.914	1304.762	0.0636
Bavarian "Total"	♂	529	50	4.8986	2.4919	79.2516	0.0528	0.2298	3.3026	−0.3026	0.4467	1363.185	1352.047	0.1006
	♀	323	50	4.1433	−0.9391	77.7815	0.0124	0.1114	4.5408	−1.5408	3.0444	1220.356	1217.221	0.0308
Bavarian "Young"	♀	238	50	3.3431	3.0329	45.6227	0.2462	0.4962	4.0820	−1.0820	2.4255	1235.504	1219.334	0.1769

and from this the distance from mean to mode was obtained by multiplying by σ.

Considering first the *skewness*, it is seen to be in all the series positive, or the mean is greater than the mode. Further we note that in all cases the value is low. Whether the values can be considered significant however can only be determined by an examination of the probable errors. The formula for the probable error of the skewness[23] is $0.67449\sqrt{\frac{3}{2n}}$. The limiting values for this probable error for values of n ranging from 197 to 529 as in the present case are respectively 0.0589 and 0.0359. Having regard to the number of cases on which the calculations are based it appears that in six out of the nine cases tabulated the skewness can be regarded as certainly or probably insignificant. In all of these six cases the skewness is less than thrice its probable error, in two cases it about equals its probable error, and in one is less. The remaining three cases out of the total (skewness, 0.1635, 0.2161 and 0.1769) are very probably or certainly significant. In general we may safely conclude, I think, that, *in the case of the weight of the brain, the distance from the mean to the mode will be very small. If the mean and mode do not coincide the mean will be greater than the mode.* This agrees with Miss Fawcett's[24] conclusion for the most important skull characters in the Naqada race. Macdonell[25] finds, however, that in the case of the English, considering the same skull characters, "if we were to draw the curves, the mean would be found in half the number of the curves to be less, and in the other half to be greater than the mode." In neither brain-weight nor skull series does there appear to be any definite preponderance in the value of the skewness of one sex over the other.

We may turn now to the other constants, which are of most significance in determining whether the distribution may be considered normal within the limits of error; viz., $\sqrt{\beta_1}$, β_2, and the criterion. The probable error of $\sqrt{\beta_1}\left(= 0.67449\sqrt{\frac{6}{n}}\right)$ ranges in value for our series between 0.0718 (n = 529), and 0.1177 (n = 197); that of $\beta_2\left(= 0.67449\sqrt{\frac{24}{n}}\right)$ between 0.1437 (n = 529) and 0.2354 (n = 197); and that of the criterion $\left(= 0.67449\right.$

[23] The formulae for the probable errors of the analytical constants are given on p. 278 of Pearson's memoir on "The mathematical theory of errors of judgment," *loc. cit. supra.*

[24] *Loc. cit.*, p. 443.

[25] *Biometrika*, vol. iii, p. 227.

$\sqrt{\frac{96}{n}}$) between 0.2873 (n = 529) and 0.4708 (n = 197). Considering the probable errors of $\sqrt{\beta_1}$, it is seen at once that of the eight "total" series three give certainly insignificant values (0.0958, 0.1139, 0.1114) for $\sqrt{\beta_1}$; two others give values which are probably insignificant (0.1694, 0.2254). One (0.2298) is probably significant; and the two remaining values (0.3623 and 0.4029) are certainly significant. The Bavarian "young" ♀ series gives a certainly significant value for $\sqrt{\beta_1}$. Taking next the deviation of β_2 from 3 in comparison with the probable error of β_2 we see that in four cases ($3 - \beta_2 = -0.1031, 0.1628, -0.1396, 0.1329$) β_2 differs from 3 by an insignificant amount. In one case (0.2036) the difference is less than twice the probable error and hence may be considered as very probably insignificant. In two cases (-0.3682 and -0.3026) the difference is less than thrice the probable error and hence may be considered possibly, or even perhaps, probably insignificant. The Bavarian ♀ series, both "total" and "young", give certainly significant values for $3 - \beta_2$. Considering finally the criterion it is seen that in all cases except the two Bavarian ♀ the criterion differs from zero by a certainly or very probably insignificant amount. These two Bavarian ♀ series differed so greatly from the normal curve in most of the analytical constants that it was thought desirable to determine their position precisely by means of another constant κ_2.[26] For the "total" series I found $\kappa_2 = 0.0032$, and for the "young" series $\kappa_2 = 0.0816$. By the scheme given by Pearson (*loc. cit.* p. 445) we see that the "total" series, when the probable errors of the constants are considered, comes very close to the condition demanding a curve of Type II ($\kappa_2 = 0, \beta_1 = 0, \beta_2$ not $= 3$). The "young" series clearly demands a curve of Type IV ($\kappa_2 > 0$ and < 1). The deviation of these Bavarian female curves from the normal type I believe to be due to an undue accumulation of individuals in one brain-weight class; viz., that from 1250 to 1300 grams. It seems altogether probable that some of the individuals which should have gone into the next higher class (represented in the "total" series by a frequency of only 26 as against 69 in the class next lower) have by some error been entered in Bischoff's lists with too low brain-weights. What the source of error was it is, of course, impossible now to determine. The abnormality of the Bavarian females has already been noted in the discussion of the means and variabilities. Leaving these two series out of account I think that on the whole we may safely conclude, as Miss

[26] $\kappa_2 = \frac{\beta_1 (\beta_2 + 3)^2}{4(\beta_2 - 3\beta_1)(2\beta_2 - 3\beta_1 - 6)}$, Pearson, *Phil. Trans.*, vol. 197, A, p. 444.

Fawcett and Macdonell (*loc. cit.* p. 443, and p. 227 resp.) have for skull characters, that:

With series of brain weighings such as are considered in this paper we shall reach for most practical purposes adequate graphical representations of the frequency by using the normal curve of deviation: $y = y_0 e^{-x^2/2\sigma^2}$.

It should always be kept in mind, however, that our series, both on the brain-weight and skull sides, are too small to fix absolutely the normality or non-normality of the variation in these characters. Some of the distributions certainly differ from normality. The conclusion stated above is to be considered simply as a *practical* result, rather than as a theoretical generalization.

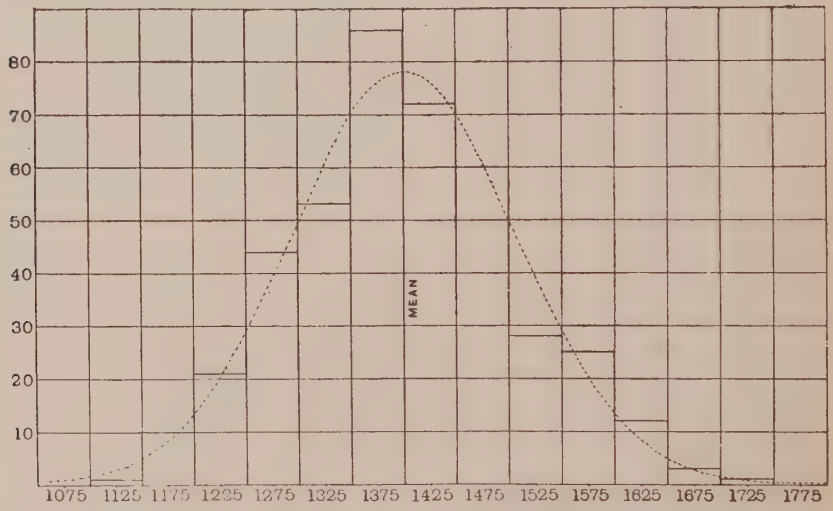


Fig. 1. Showing the Distribution of Variation in the Brain-weight of Adult Swedish Males

This result seems to be of considerable importance as indicating the worth of brain-weight statistics. It shows that such statistics justify careful study and analysis, and that, contrary to the statements of certain recent writers on the subject, there is no general fallacy inherent in the data themselves which renders abortive any attempt to reach through them the truth regarding the mass relations of the brain.

In order to test exactly how well the normal curve represents the data in a single case I have fitted the Swedish male "total" series with a normal curve. The frequency histogram and its fitted curve are shown graphically in the accompanying diagram. The unit of x is 50 grams. The scale of frequency at the left of the diagram gives y.

The general equation $y = y_0 e^{-x^2/2\sigma^2}$, where in this case $y_0 = \frac{416}{\sigma\sqrt{2\pi}}$ and $\sigma = 2.1266$, becomes $y = 78.0401\ e^{-0.01106x^2}$, with the origin at 1400.481. Applying Pearson's test of the goodness of fit,[27] and comparing *areas* instead of mid-ordinates, as is necessary where the number of frequency groups is so small, we get the results shown in the accompanying table. The table gives the observed frequencies (m_r'),

GRAMS OF BRAIN-WEIGHT	OBSERVED	CALCULATED	$\frac{(m_r - m_r')^2}{m_r}$
Under 1100	0	0.981	0.981
1100–1150	1	2.9	1.24
1150–1200	10	8.5	0.26
1200–1250	21	20.3	0.02
1250–1300	44	39.0	0.64
1300–1350	53	60.4	0.91
1350–1400	86	75.2	1.55
1400–1450	72	75.3	0.14
1450–1500	60	60.8	0.01
1500–1550	28	39.4	3.29
1550–1600	25	20.6	0.94
1600–1650	12	8.7	1.25
1650–1700	3	2.9	0.003
1700–1750	1	0.8	0.05
1750 and over	0	0.036	0.036
Totals.....	416	415.817	11.320

the calculated frequencies (m_r), and the ratio of the squared difference between the two to the calculated $\left(\frac{(m_r - m_r')^2}{m_r}\right)$.

Here n' the number of frequency groups is 15 and $\chi^2 = 11.32$. From Elderton's Tables[28] the value of P may be obtained. With $n' = 15$ and $\chi^2 = 11$ the value of P as given in the table is 0.686036. Or, expressed in words, if the brain-weight of Swedish males obeyed the "normal" distribution of freuenqcies we should expect to get a greater divergence between theory and observation in roughly 65 out of every 100 trials when the trials were based on random samples of 416 individuals each. In other words, the fit may be considered very fair, and certainly entirely satisfactory for all practical purposes.

[27] *Phil Mag.*, vol. l, pp. 157–175.

[28] *Biometrika*, vol. i. p. 161

VII. THE CORRELATION OF BRAIN-WEIGHT WITH OTHER CHARACTERS

In the introductory portion of this chapter it was stated that one of the chief objects for which the work was undertaken was to measure exactly the degree and nature of the association between brain-weight and other characters in man. All workers on the subject have realized the importance of such determinations and numerous attempts have been made to arrive at them. The methods used, however, have not been such as to lead to definite and sound conclusions. These methods have in the main consisted in tabulating the mean brain-weights for various groupings of the other characters under consideration. In this way it can of course be determined whether there is any shift of the mean as the other character changes. Further than this one cannot go. Besides, in this method there lurk numerous pitfalls unless one uses it with a clear understanding of some of the fundamentals of statistical science. Different results can be obtained as the material is differently grouped. Hence, it is not strange that we find conservative anatomists making only very general statements as to the correlation of brain-weight with other characters. and, in those cases where an attempt at greater precision of statement is made, considerable difference of opinion as to what conclusions shall be drawn from the data. What are wanted in the case are not simply inspections and general "appreciations" of tabular lists of brain-weighings, or of ratios and indices, or even of correlation tables or regression lines, but instead definitely determined coefficients of correlation with their probable errors. With these we can get in given cases precise statements of the degree of correlation between different characters.

It would of course be highly desirable to measure the correlation between the weight of the brain and a great variety of other organs and characters of the body. Such characters will at once suggest themselves to anyone interested in the problems of correlation. For instance, aside from age, stature and body-weight for which material is available, there are the whole range of skull characters; the weights of the various viscera; the weight and volume of the spinal cord; characters of the muscular system, etc. A study of the correlation between any or all of these characters and the weight of the brain would be most interesting. But, unfortunately, material for such study cannot be had. Brain-weight statistics usually provide data for but few other characters; in the majority of cases only sex, age and stature. In two of the series used in this work, viz., the Swedish and Hessian, this was the case. For these two groups I have determined all possible correlations for both sexes, and for two age group-

ings ("young" and "total"). This gives as the pairs of characters, (*a*) brain-weight and stature, (*b*) brain-weight and age, (*c*) stature and age. Bischoff's Bavarian material furnished, in addition to the above, data on the body-weight. The following correlations have been determined for this material: (*a*) brain-weight and age ("total" and "young"), (*b*) brain-weight and stature ("total"), (*c*) brain-weight and body-weight ("total"), (*d*) body-weight and stature ("total"). In the case of the Bohemian material data were furnished from which the correlation between skull length and skull breadth could be determined so that for this group I have been able to calculate coefficients of correlation for the following pairs of characters: (*a*) brain-weight and stature ("young"), (*b*) brain-weight and age ("total"), (*c*) brain-weight and skull length ("young"), (*d*) brain-weight and skull breadth ("young"), (*e*) skull length and skull breadth ("young"). In table 9 are exhibited the coefficients of correlation together with their probable errors. References are given in table 9 telling for each coefficient the original table from which it was calculated. The coefficients of correlation were determined from the usual Bravais formula according to which the coefficient of correlation

$$r = \frac{S\,(xy)}{N\sigma_1\sigma_2},$$

where x and y are deviations from the means of the two correlated characters and σ_1 and σ_2 are the respective standard deviations; the usual[29] method of evaluating r from the above equation was used.

The most striking fact of a general nature shown by this table is the generally low degree of correlation which exists between the weight of the brain and other characters. The coefficients run noticeably lower even than those of skull characters (*cf.* Macdonell's Table V[30]) and very markedly below those between different characters of the long bones.[31] On the other hand our brain-weight correlations give values of the same general order of magnitude as those found by Greenwood[32] for various abdominal and thoracic viscera. Attention may be called here to the general uniformity of the correlations for the same characters in different series. All the brain-weight and stature correlations are seen to be positive, while with a single exception (Bavarian female "young" series) all the correlations of brain-weight with age are negative. The coefficients are generally

[29] Yule, *Jour. Roy. Stat. Soc.*, vol. lx, part iv, pp. 1–44 (Reprint).
[30] *Biometrika*, vol. iii, p. 232.
[31] Lee and Pearson, *Phil. Trans.*, vol. 196, A, pp. 228, 229.
[32] *Loc. cit.*, p. 19 above.

TABLE 9

Brain-weight. Correlations

CHARACTER AND SERIES	SWEDES ♂ No.		Table	SWEDES ♀ No.		Table	HESSIANS ♂ No.		Table	HESSIANS ♀ No.		Table
Brain-weight and Stature (Total)	416	0.1830±0.0320	1	233	0.3490±0.0388	2	475	0.1823±0.0299	13	281	0.1828±0.0389	14
Brain-weight and Stature (Young)	262	0.1796±0.0403	3	127	0.3390±0.0530	4	291	0.1741±0.0383	15	173	0.1809±0.0496	16
Brain-weight and Age (Total)	416	−0 2493±0.0310	5	233	−0.2336±0.0418	6	475	−0.1673±0.0300	17	281	−0.3598±0.0350	18
Brain-weight and Age (Young)	262	−0.1705±0.0405	7	127	−0.1512±0.0585	8	291	−0.0750±0.0393	19	173	−0 1650±0 0499	20
Stature and Age (Total)	416	−0.1620±0.0322	9	233	−0.2279±0.0419	10	475	0.0'81±0.0308	21	281	−0.0915±0.0399	22
Stature and Age (Young)	262	−0.2549±0.0390	11	127	−0.2120±0.0572	12	291	0.2102±0.0378	23	173	−0.0474±0.0512	24

CHARACTER AND SERIES	BOHEMIANS ♂ No.		Table	BOHEMIANS ♀ No.		Table	BAVARIANS ♂ No.		Table	BAVARIANS ♀ No.		Table
Brain-weight and Stature (Total)							365	0.1664±0.0343	35	241	0.2236±0.0413	36
Brain-weight and Stature (Young)	266	0.2034±0.0397	25	133	0.2168±0.0557	26						
Brain-weight and Age (Total)	372	−0.2045±0.0335	27	197	−0.2558±0.0449	28	529	−0.1225±0.0290	37	323	−0.2405±0.0354	38
Brain-weight and Age (Young)							365	−0.0100±0.0353	39	238	0.0114±0.0412	40
Brain-weight and Skull Length (Young)	299	0.5482±0.0273	29	159	0.3604±0.0465	30						
Brain-weight and Skull Breadth (Young)	299	0.4655±0.0306	31	159	0.5041±0.0399	32						
Brain-weight and Body-weight (Total)							365	0.1671±0.0343	41	241	0.2260±0.0412	42
Skull Length and Skull Breadth (Young)	299	0.4006±0.0327	33	159	0.1954±0.0514	34						
Body-weight and Stature (Total)							365	0.3962±0.0298	43	241	0.4334±0.0352	44

lower for the "young" than for the "total" series, as is to be expected. In the case of the correlation of brain-weight with age, some of the coefficients for the "young" series are evidently insignificant in comparison with their probable errors. The correlations between stature and age are negative with the exception of the Hessian male series. The positive sign in these series arises from the fact that in this material the age class 15 to 20 was included. All growth in stature has not stopped at age 20, and as a consequence there are included in the tables (21 and 23) five individuals of unusually small stature and low age. These serve to change the sign of the coefficients. Their greater effect in the "young" series is apparent. The detailed discussion of the various brain-weight correlations and the regressions based on them I propose to take up in separate sections now to follow.

VIII. BRAIN-WEIGHT AND SEX

All brain-weight statistics show that the brain of the male is absolutely heavier than that of the female. In the series here discussed the absolute differences in mean brain-weight between males and females are as follows:

	MALE MEAN—FEMALE MEAN	
	"Total" series	"Young" series
Swedes	147.8	145.8
Bohemians	143.9	146.2
Bavarians	142.8	133.6
Hessians	131.9	125.6

From this table the following points are to be noted:

a. Considering the size of the probable errors involved it is evident at once that the *absolute* difference is sensibly the same for all four races. Taking the extremes of the "total" column the difference between the Swede and Hessian sex differences is 15.9 with a probable error of $\pm$ 7.9, or in other words the difference is almost exactly twice its probable error and cannot be considered certainly significant. The "young" series points to the same conclusion. So then, Weisbach's law for stature that the greatest sex differences occur in those races having the highest mean stature, does not appear to hold for brain-weight, so far as absolute differences are concerned.

b. The absolute sex differences are sensibly the same for both age groupings. We may conclude then, that whatever changes occur in the weight

of the brain with advancing age act in such a way as to leave the absolute sex difference unchanged.

These absolute differences may be converted into relative differences by expressing them as percentages of the ♂ and ♀ means. When so expressed they take the following form:

Sex differences in relation to means

RACE	"TOTAL" SERIES		"YOUNG" SERIES	
	Percentage of ♂ mean	Percentage of ♀ mean	Percentage of ♂ mean	Percentage of ♀ mean
	per cent	*per cent*	*per cent*	*per cent*
Swedes	10.6	11.8	10.3	11.5
Bavarians	10.5	11.7	9.8	10.8
Bohemians	9.9	10.97	10.0	11.1
Hessians	9.5	10.5	8.9	9.8

The relative differences confirm the conclusions reached from the examination of absolute differences. The result that the changes which take place in the weight of the brain during adult life affect both sexes to an equal degree relatively seems to be of some importance. It implies that the factors which bring about the gradual lowering in the weight of the brain with advancing age are organic in a strict sense, and not dependent on environmental conditions.

The same thing is shown if the relative differences expressed in another way, namely by taking the ratio of male to female mean, are examined. For our series these ratios are as follows:

Sex ratios. Means. Brain-weight

RACE	"TOTAL" SERIES	"YOUNG" SERIES
Swedes	1.118	1.115
Bavarians	1.117	1.108
Bohemians	1.110	1.111
Hessians	1.105	1.098

The sex ratios for a number of other characters are given in the following table for comparison. The values in this table are taken from a table in Pearson's *Chances of Death*.[33]

[33] Vol. i, p. 374

Sex ratios. Means. Various physical characters

ORGAN OR CHARACTER	SEX RATIO
Body weight, Babies	1.034
Body weight, Children	1.038
Body weight, Adults	1.193
Weight of Vital Organs	1.130
Stature, Children	1.007
Stature, Adults	1.077
Height, Sitting	1.032
Long Bones	1.086
Chest Girth	1.024
Squeeze of Hands	1.207
Keenness of Sight and Touch	1.061
Skull Capacity	1.124
Skull Circumference	1.042
Cephalic Index	0.997
Head Index	0.995
Profile Angle	0.994
Alveolar Angle	0.994
Nose and Palate	1.013

We may turn next to the question of the relative variability of the sexes in respect to brain-weight. Pearson[34] found that in the case of the English, the female showed "slightly more" variability than males; the French data which he worked over gave sensibly equal variabilities for the sexes, as did also Bischoff's Bavarian data. In the following table are shown the differences between the male and female coefficients of variation with the probable errors of the differences. The values tabulated are the male minus the female constant in each case.

Relative variability of the sexes. Male—Female

RACE	COEFFICIENT OF VARIATION	
	"Total" series	"Young" series
Bohemians	0.427±0.320	0.740±0.375
Hessians	−0.029±0.293	−0.036±0.366
Bavarians	−0.222±0.254	0.546±0.305
Swedes	−0.451±0.310	−0.600±0.422

In three cases out of the eight the male has a larger coefficient of variation, but in no instance can the differences between male and female coefficients be considered significant when compared with the probable errors.

[34] *Chances of Death*, vol. i, pp. 321, 322.

In only one case (Bohemian "young" series) does the difference approach closely to a value even twice as great as its probable error. Therefore we must conclude that, so far as the series here considered are concerned, there is no significant difference between males and females in respect to variability in the weight of the brain. In view of the general reliability, from a statistical standpoint, which has been previously demonstrated to exist in the material on which this work is based, it seems not unreasonable to predict that it will be found that equal variability of the sexes in brain-weight holds generally.

Another problem which presents itself in this connection is as to how much of the observed difference between males and females in brain-weight is to be accounted for by the fact that the male body is in general larger than the female. In other words, would a group of females of the same stature and age, say, as a given group of males have the same brain-weight? It is possible to reach a general solution of this problem by the use of characteristic equations based on the correlations and regressions. I propose now to discuss this question by this method. Of course, the prediction may be made by characteristic equations in either direction; *i.e.*, we may predict the probable brain-weight of a group of males having other characters the same as in a given group of females, or we can predict the probable brain-weight of a group of females having the characters the same as in a given group of males. In the former case characteristic equations based on the male data would be used, in the latter the equations would be those deduced from female data. We may consider first the characters, age, and stature. In the following tables are given (*a*) the probable brain-weights of a group of individuals of the sex indicated, as calculated from the characteristic equation referred to in the last column of the table; (*b*) the observed brain-weights of a group of the opposite sex having for the other characters the mean values used in the characteristic equation in calculating (*a*); (*c*) the difference between (*a*) and (*b*); (*d*) a reference to the equation from which (*a*) is calculated.

The mean difference here is 97. The mean observed difference between male and female without any allowance for stature and age is 137.8 for the same races. So then the differences in stature and age between the males and females in our Swedish and Hessian samples account for only 40.5 grams or about 29 per cent of the observed difference in mean brain-weight between the sexes.

Let us now proceed to the reversed prediction, and find the probable brain-weights of a group of males having the same stature and age as the means for these characters in the Hessian and Swedish females. The

results are shown in table 11, which is arranged in the same manner as the preceding table.

The mean difference here is 107 or in other words we reach essentially the same result as before. The general conclusion up to this point then is that there is a difference in the mean brain-weight between the sexes of roughly 100 grams after allowance has been made for stature and age differences by taking the mean brain-weight of an array of individuals of one sex having for type of stature and age the respective means of these characters observed in the other sex. Differences in stature and age account for less than one-third of the observed sex difference in brain-weight.

TABLE 10

Probable female brain-weight with stature and age equal to observed male

RACE		PREDICTED FEMALE BRAIN-WEIGHT	OBSERVED MALE BRAIN-WEIGHT	DIFFERENCE	EQUATION
Hessian	(Total)	1288	1392	104	25
	(Young)	1309	1406	97	26
Swede	(Total)	1307	1400	93	21
	(Young)	1320	1415	95	22

TABLE 11

Probable male brain-weight with stature and age equal to observed female

RACE		PREDICTED MALE BRAIN-WEIGHT	OBSERVED FEMALE BRAIN-WEIGHT	DIFFERENCE	EQUATION
Hessian	(Total)	1358	1260	98	23
	(Young)	1369	1280	89	24
Swede	(Total)	1371	1253	118	19
	(Young)	1392	1269	122	20

We may next determine what part of the observed sex difference in brain-weight is to be accounted for by differences in skull length and breadth. Here only one race, the Bohemian, can be used on account of lack of material. Let us examine the effect on the mean brain-weight of selecting a group of individuals of one sex having the same mean skull length and skull breadth as the opposite sex. Making the designated selection first from the males and then from the females we have the results shown in table 12.

The mean of the two differences is 75, while the observed difference between male and female mean brain-weights in the Bohemian "skull"

series is 142 or in other words by selecting from either sex individuals with mean skull length and skull breadth equal to those found in the opposite sex, we are able to reduce the sex difference in mean brain-weight by about 67 grams, or 47 per cent. This result is of considerable interest. We can come nearer to the brain-weight of one sex by selecting skull length and breadth to the means of that sex than we can by selecting stature and age, but still not a great deal nearer. We should on general grounds expect that aside from skull capacity, skull length and skull breadth would be the two characters whose selection would indirectly modify brain-weight the most. Admitting this to be the case there still remains a considerable difference to be accounted for. It is interesting to note that the sexes are brought nearer together in brain-weight when males are selected than when the selection is from the females. This is explained by the fact that

TABLE 12

Probable male brain-weight with mean skull length and skull breadth equal to the observed female

RACE	PREDICTED MALE BRAIN-WEIGHT	OBSERVED FEMALE BRAIN-WEIGHT	DIFFERENCE	EQUATION
Bohemian	1396	1311	85	35

Probable female brain-weight with mean skull length and skull breadth equal to the observed male

RACE	PREDICTED FEMALE BRAIN-WEIGHT	OBSERVED MALE BRAIN-WEIGHT	DIFFERENCE	EQUATION
Bohemian	1390	1455	65	36

the brain-weight is somewhat more highly correlated with the skull characters in the males in our series.

Finally, we may examine the effect of a selection of stature and body-weight on the sex difference in brain-weight. The material here comes from the Bavarian series, and the prediction is made in the same way as in the other cases; viz., by selecting males with mean stature and body-weight equal to the female means and *vice versa*, and determining the mean brain-weight of the selected groups. The results are shown in table 13.

Here the better result is obtained by selecting from the females on account of the higher correlation between brain-weight and body-weight and brain-weight and stature in this sex. The mean of the two differences is 99, and the observed sex difference in the case is 139 grams. So then

the differences between the sexes in body-weight and stature account for 39 grams or 28 per cent of the observed difference in mean brain-weight. In other words, by selecting individuals on the basis of body-weight and stature alone we shall produce about the same degree of change in the mean brain-weight as by selecting on the basis of stature and age alone.

Taking all the results together we reach the general conclusion that the difference between the sexes in mean brain-weight is only in part to be accounted for by differences in other bodily characters. While in general it is true that a small body has a brain of low weight, yet in order for the observed difference in mean brain-weight in men and women to be due to this factor alone either the women ought to have very much smaller bodies than they actually possess, or the men ought to have larger bodies, or both sexes ought to be different from what they actually are in size of body in the directions indicated. Of course it is easy to say by way of

TABLE 13

Probable male brain-weight with mean stature and body-weight equal to the observed female

RACE	PREDICTED MALE BRAIN-WEIGHT	OBSERVED FEMALE BRAIN-WEIGHT	DIFFERENCE	EQUATION
Bavarian	1321	1219	105	29

Probable female brain-weight with mean stature and body-weight equal to the observed male

RACE	PREDICTED FEMALE BRAIN-WEIGHT	OBSERVED MALE BRAIN-WEIGHT	DIFFERENCE	EQUATION
Bavarian	1261	1357	96	30

speculation that the smaller brain of women is due to the fact that the female human organism is subjected to less strenuous demands along the lines of motor and intellectual activity than the male. But such speculation leads to nothing on account of our lack of definite scientific evidence as to the degree of correlation between the weight of the brain and amount and intensity of psychic activity.

One other question remains to be considered in connection with the relation of brain-weight to sex. Does either sex show any considerably higher degree of correlation between brain-weight and other characters than the other? Table 9 furnishes data on this point. With the exception of the Swedish brain-weight and age correlations and the brain-weight and skull-length correlations it is seen that the female coefficients are uniformly larger than the male. The differences are in many of the cases not significant in comparison with their probable errors but the general

tendency towards higher correlations in the females is clearly evident. The meaning of this tendency is in this case difficult to conjecture. It falls in line with the result which has been pointed out by Lee and Pearson in several papers,[35] viz., that for physical characters generally women usually show a higher degree of correlation than men, in civilized races.

IX. BRAIN-WEIGHT, AGE AND STATURE

The coefficients of correlation between brain-weight and age and stature respectively have already been given in table 9, but before proceeding further in the discussion of their significance it is necessary to determine whether the regressions are linear. One might on general grounds expect the brain-weight and age correlations to be skew, since this is generally the case with growth correlations where one of the variables is age and the other the absolute magnitude of some organ or character. If in any case the correlation turns out to be skew, it of course greatly complicates the problem.

The method of analysis which I have followed in determining the degree of approach to linearity of the regressions is that given by Pearson.[36] The two constants which are of the greatest significance here are, (*a*) the mean square deviation of the means of the arrays from the regression line, Σ_M^2, and (*b*) a constant η, called the correlation ratio, giving the mean reduction in variability of an array as compared with the whole population. Evidently

$$\Sigma_M^2 = \sigma_M^2 - r^2\sigma^2 \quad \ldots\ldots\ldots\ldots\ldots\ldots \text{(i)},$$

and

$$\eta = \frac{\sigma_M}{\sigma} \quad \ldots\ldots\ldots\ldots\ldots\ldots\ldots \text{(ii)},$$

whence by simple substitution $\Sigma_M^2 = (\eta^2 - r^2)\,\sigma^2$ ..... (iii),

where in these equations σ is, as usual, the standard deviation of the variates about the mean for the whole population, r is the coefficient of correlation between the two variables concerned, and σ_M is the standard deviation of the means of the arrays about the mean of these means. The deviation

[35] *E.g.*, On the relative variability and correlation in civilized and uncivilized races, *Roy. Soc. Proc.*, vol. lxi, pp. 343–357.

[36] *Roy. Soc. Proc.*, vol. lxxi, pp. 303–313, especially the footnote, pp. 303, 304. Since the above was written a very full treatment of the whole subject of skew correlation and non-linear regression by Prof. Pearson has appeared as Mathematical contributions to the theory of evolution, XIV, *Drapers' Company Research Memoirs*, Biometric Series II, 1905.

of Σ_M from zero, and of η from r, measure the deviation of the system from linearity.[37]

I propose to discuss the relation of brain-weight first to age alone, then to stature alone, and finally to both age and stature together.

In table 14 are exhibited the values of Σ_M and η for the correlations between brain-weight and age discussed in this paper. In calculating σ_M from which to obtain η according to the relation given above, in this and all other cases the means of the arrays were weighted with the number of cases on which they were based. This procedure of course gave the

TABLE 14

Analytical constants for linearity of regression. Brain-weight and age

RACE AND SERIES	r		η		Σ_M	
	♂	♀	♂	♀	♂	♀
Swedish:						
Total.....	-0.2493 ± 0.0310	-0.2336 ± 0.0418	0.2876	0.2770	0.1434σ	0.1489σ
Young.....	-0.1705 ± 0.0405	-0.1512 ± 0.0585	0.2251	0.2143	0.1469σ	0.1519σ
Hessian:						
Total.....	-0.1673 ± 0.0300	-0.3598 ± 0.0350	0.2002	0.3864	0.10996σ	0.1409σ
Young.....	-0.0750 ± 0.0393	-0.1650 ± 0.0499	0.1411	0.1961	0.1195σ	0.1060σ
Bavarian:						
Total.....	-0.1225 ± 0.0290	-0.2405 ± 0.0354	0.1962	0.3481	0.1533σ	0.2517σ
Young.....	-0.0100 ± 0.0353	0.0114 ± 0.0412	0.0676	0.1958	0.0669σ	0.1955σ
Bohemian:						
Total.....	-0.2045 ± 0.0335	-0.2558 ± 0.0449	0.2441	0.3033	0.1333σ	0.1628σ

mean of the means of the arrays the same value as the general population mean calculated from the elemental frequency distribution.

It is seen at once that η differs from r and Σ_M from zero in all cases. This, of course, implies departure of the system from linearity. But evidently

[37] Of course the deviation of η from r must be considered numerically simply, because η is necessarily a positive quantity from (ii) above, since neither σ_M nor σ can be negative. If the difference between η and r were taken with reference to sign in cases where r is minus, an altogether false notion of the degree of departure of the regression from strict linearity would be obtained. The degree of this departure will always, of course, be immediately given by Σ_M whatever the sign of r. Blakeman's (*Biometrika*, vol. 4, pp. 332–350, 1905) test for linearity of regression appeared later than the present study. Nowadays it is possible to discuss the point with greater mathematical elegance, but in this particular case the conclusion would be in nowise altered.

deviations of a system from linearity may be due to either one or both of two causes. Either, on the one hand, the system may be truly non-linear, in which event the means of the arrays will be fitted better by some curve than by a straight line, or, on the other hand, the points fixed by the means of the arrays may not lie exactly on a straight line and still no curve will represent the relationship between the two variables concerned better than a straight line so drawn that the mean square of the deviations of the points from the line is a minimum. In the first case we have true non-linearity of the regression, while in the second the deviation from linearity is due to the errors of random sampling, and it might reasonably be expected that if the whole population could be studied the regression would become strictly linear. Now evidently in both these cases η will differ from r and Σ_M from zero, so that recourse must be had to some further method in order to determine into which class a given case falls. Two such methods immediately suggest themselves: one, to examine the probable errors involved, the other, to inspect the fitted regression line.

An examination of table 14 makes it immediately evident that the differences between η and r (without regard to sign) give values of the same order of magnitude as the probable errors of r. In only one case is this difference as great as three times the probable error of r, and in the great majority of cases it does not approach such a value. So then it seems probable that in our series the regression of brain-weight on age is linear within the errors arising from random sampling. The approach to linearity is sensibly the same both for the whole period of adult life and for the younger half of this period.

In order to bring out the facts graphically I have had prepared a series of diagrams showing the regression lines for brain-weight on age. For the sake of economizing space, and since there is essential agreement between the different series with respect to η, it was decided not to publish all the regression diagrams. I have chosen for representation here the regression lines for the Swedish and the Hessian data. These are shown in figures 2 to 9.

These diagrams show clearly, I think, that the regression of brain-weight on age in these cases is sensibly linear within the ages 15 to 80, so far as can be judged on the material available. The regression diagrams for the other two racial groups show the same relation. Obviously no simple curve will represent the systems of points shown in these diagrams 2 to 9 better than the straight line does. So then, until we have very much larger samples of material to work with, we shall be justified in assuming for practical purposes linearity of regression between brain-

weight and age, and in considering the deviations of the means of the arrays from the regression line to be due to errors incident to sampling in lots of less than 500 individuals.

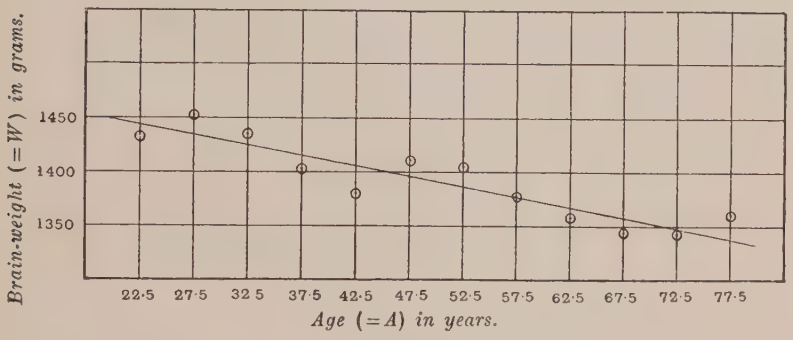


FIG. 2. PROBABLE BRAIN-WEIGHT FOR GIVEN AGE. SWEDISH ♂ TOTAL

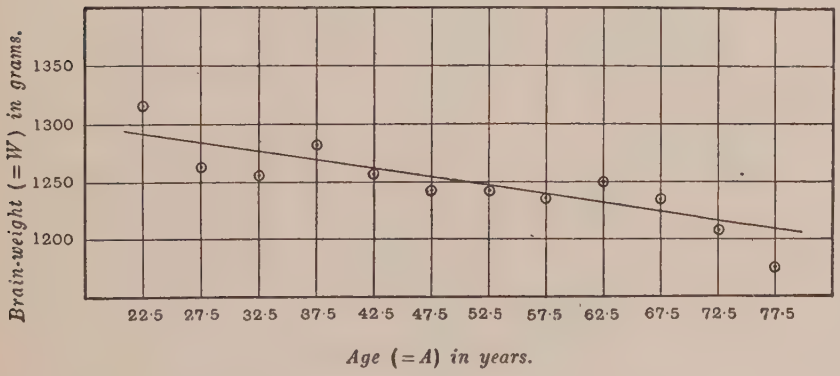


FIG. 3. PROBABLE BRAIN-WEIGHT FOR GIVEN AGE. SWEDISH ♀ TOTAL

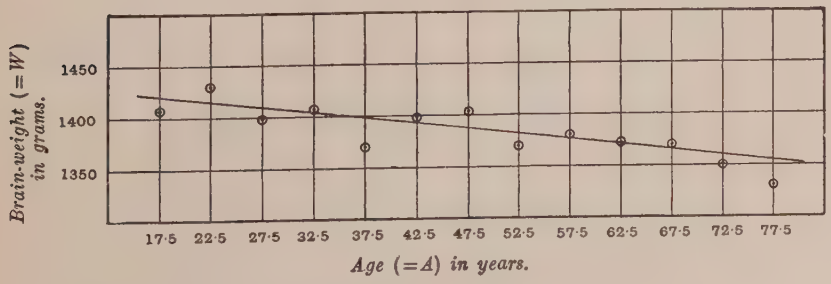


FIG. 4. PROBABLE BRAIN-WEIGHT FOR GIVEN AGE. HESSIAN ♂ TOTAL

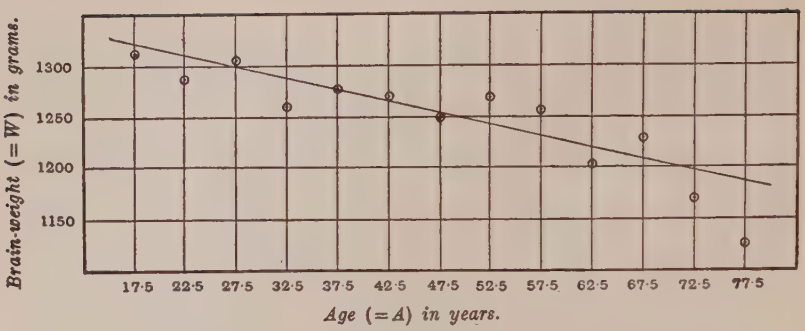


FIG. 5. PROBABLE BRAIN-WEIGHT WITH GIVEN AGE. HESSIAN ♀ TOTAL

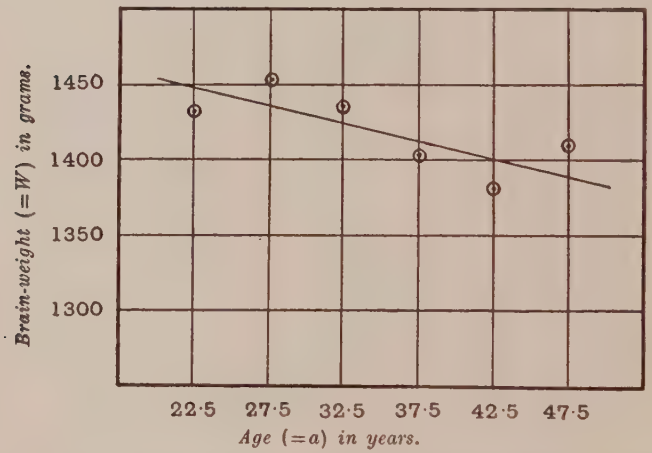


FIG. 6. PROBABLE BRAIN-WEIGHT WITH GIVEN AGE. SWEDISH ♂ YOUNG

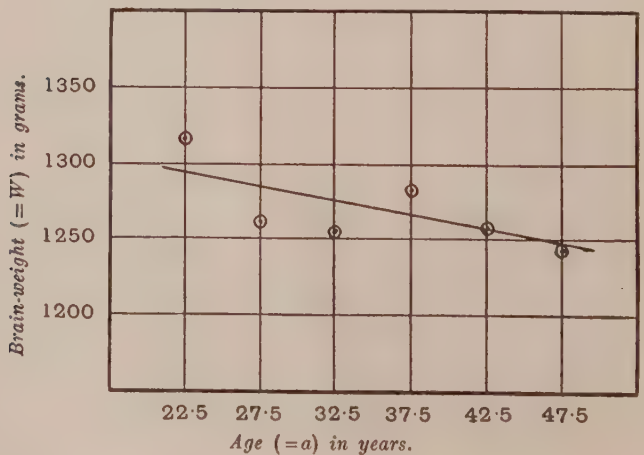


FIG. 7. PROBABLE BRAIN-WEIGHT WITH GIVEN AGE. SWEDISH ♀ YOUNG

The equations to the regression lines for these diagrams may now be given. For convenience in practical use I have put these equations in the form of "characteristic" equations. The significance of the letters used is as follows: W denotes the probable brain-weight in grams of an array of individuals of age type A or a. The ages included by A are 15 or 20 to 80; by a 15 or 20 to 50, or in other words the equations containing A are deduced from the "total" series and those containing a from the

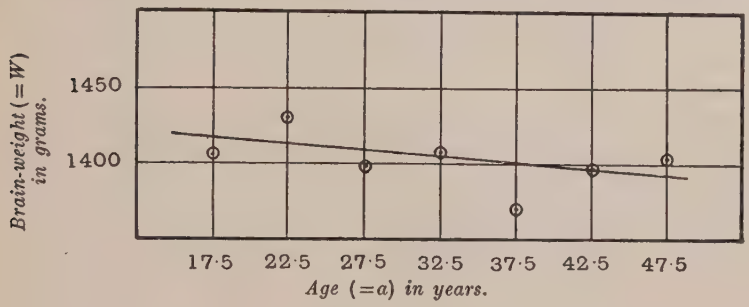


FIG. 8. PROBABLE BRAIN-WEIGHT FOR GIVEN AGE. HESSIAN ♂ YOUNG

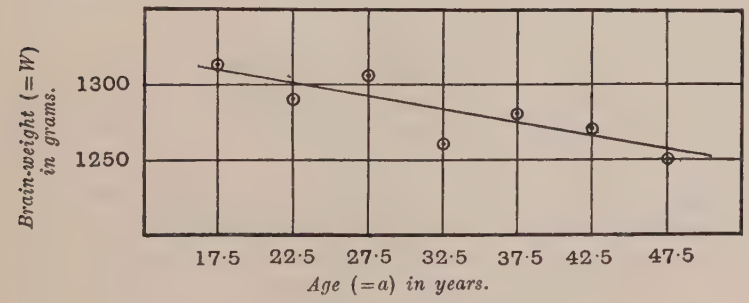


FIG. 9. PROBABLE BRAIN-WEIGHT FOR GIVEN AGE. HESSIAN ♀ YOUNG

"young" series. Σ is the standard deviation of the array having the probable W as its mean.

(1) Swedish ♂ $W = 1487.783 - 1.939\,A$, $\Sigma = 102.972$
(2) Swedish ♂ $W = 1501.411 - 2.372\,a$, $\Sigma = 107.582$
(3) Swedish ♀ $W = 1326.475 - 1.549\,A$, $\Sigma = 97.969$
(4) Swedish ♀ $W = 1340.438 - 1.988\,a$, $\Sigma = 104.329$
(5) Hessian ♂ $W = 1439.734 - 1.119\,A$, $\Sigma = 111.087$
(6) Hessian ♂ $W = 1432.497 - 0.847\,a$, $\Sigma = 111.014$
(7) Hessian ♀ $W = 1361.067 - 2.259\,A$, $\Sigma = 95.512$
(8) Hessian ♀ $W = 1339.298 - 1.735\,a$, $\Sigma = 100.445$

The foregoing constants bring out a number of interesting and important facts regarding the relation of brain-weight to age. In the first place it is clear that diminution in brain-weight with advancing years takes place very slowly. In 10 years the Swedish males lose 19.39 grams and the Hessian males 11.19 grams.[38] These are comparatively speaking slight changes, amounting respectively to 1.3 per cent and 0.8 per cent of the mean brain-weight for the whole adult period. In the same length of time (10 years) males lose an average of 0.34 inches in stature or approximately 0.5 per cent of the mean stature.[39] The diminution in brain-weight seems thus to be relatively somewhat more rapid than the diminution in stature.

The fact that the regression is linear implies that this diminution of brain-weight with advancing age so far as can be judged from the material here discussed is sensibly equally distributed over the whole adult period. To be sure the slope of the lines is in all but one case (the Swedes) less when the individuals between 20 and 50 are treated separately, but the difference is a small one when the probable error is considered (*cf.* table 9 or 14). Splitting the material up in this way of course greatly reduces the already statistically small series, so that the probable errors of the coefficients of correlation for the "young" series become large. It is significant, however, that with a single exception (the otherwise abnormal Bavarian female series) all the coefficients of correlation between brain-weight and age for the "young" series are negative. The evidence presented by each of the four series here discussed, which are representative and have been shown to be statistically trustworthy, and which if not as large as might be desired by the statistician are among the largest ever collected by the neurologist, is mutually accordant, and leads to but one conclusion: namely, that *after age 15 to 20 there is a steady though very gradual diminution in the weight of the brain with advancing age.* This conclusion I have emphasized because it is not in agreement with that reached by some eminent neurological authorities. I would further emphasize the fact that the conclusion is by no means final in the mind of the biometrician. It simply marks the point to which the material at present available leads.

[38] In order to economize space I have not tabulated the regression coefficient separately. It will be understood of course by anyone wishing to refer to or use these regression coefficients that the number prefixing a letter in the second member of any of the characteristic equations 1 to 36 is the regression coefficient (either gross or partial) of brain-weight on the character indicated by the letter.

[39] Powys, A. O., Data for the problem of evolution in man. Anthropometric data from Australia, *Biometrika*, vol. i, pp. 30–49.

Very much larger series of brain-weighings are needed before the precise form of the regression can be finally determined. But the fact that four distinct series of fair length taken by different observers on different races lead to such closely accordant results as those shown in table 14 may be taken as very strong presumptive evidence of the essential correctness of the conclusion reached.

It is noteworthy that there is no tendency towards more rapid diminution in brain-weight after age 50 to 60. The rate of diminution shows no such marked changes at this time as has been maintained to occur by many of the workers on the subject.

With the exception of the Swedish series the females in all cases show a higher correlation between brain-weight and age than the males. This difference seems in the case of the Hessians and Bavarians (total series) to be probably significant. According to Powys' (*loc. cit.*) figures the slope of the regression line between stature and age is slightly steeper for the females. It is difficult to assign any reason for this greater correlation between brain-weight and age in the female. It has been shown above that there is in general no significant difference between the sexes in respect to variability in brain-weight, so that the usual formula of "low variation and high correlation" connoting stability of type does not hold in this case. It is possible that we have here an expression in a particular case of a greater general "evenness" in the females of the bodily changes accompanying increasing age, which in turn might be due to the generally more even environmental conditions to which women are subjected. It is noteworthy in this connection that the correlation in respect to duration of life is generally higher between pairs of female relatives than between pairs of male relatives.[40]

The linearity of the regression of brain-weight on age is of interest as possibly indicating a fundamental difference in the modes of action of the biological processes of growth on the one hand and senescence on the other. When growth in absolute magnitude of a character is plotted on a base line of age the result is usually a curved line (*cf.* for example the 12 to 25 portion of Powys' stature curve, *loc. cit.*), which implies, of course, that the amount of increment in the character in question is not the same for each unit of time. On the other hand in the case of decrease of brain-weight and stature with advancing age the decrement seems to be practically uniform for each unit of time. If these relations should prove to be generally true they would furnish a very interesting field for further study and analysis.

[40] Beeton and Pearson, *Biometrika*, vol. i, p. 60.

Turning now to the relation of brain-weight to stature, we have in table 15 the values of r, η and Σ_M, for all the brain-weight and stature correlations.

Again, the values of η and Σ_M are seen to diverge considerably from r and 0 respectively. It is clear that on the whole the regression of brain-weight on stature approaches less closely to linearity than does the regression of brain-weight on age. This is, I think, a somewhat remarkable result, and one not likely to have been foreseen.

In order to determine whether the departure of the regression from strict linearity is due simply to errors of sampling or to a fundamentally different law of relation between the two variables it is again necessary to

TABLE 15

Linearity of regression. Brain-weight and stature

RACE AND SERIES	r		η		Σ_M	
	♂	♀	♂	♀	♂	♀
Swedish						
Total.....	0.1830±0.0320	0.3490±0.0388	0.2439	0.3847	0.1612σ	0.1618σ
Young.....	0.1796±0.0403	0.3390±0.0530	0.2837	0.4738	0.2196σ	0.3310σ
Hessian						
Total.....	0.1823±0.0299	0.1828±0.0389	0.2864	0.3215	0.2209σ	0.2645σ
Young.....	0.1741±0.0383	0.1809±0.0496	0.2714	0.3807	0.2082σ	0.3350σ
Bavarian						
Total.....	0.1664±0.0343	0.2236±0.0413	0.2262	0.3270	0.1532σ	0.2386σ
Bohemian						
Young.....	0.2034±0.0397	0.2168±0.0557	0.2419	0.3591	0.1309σ	0.2863σ

have recourse to regression diagrams. I have selected eight diagrams which are fairly representative.

It is evident from these diagrams that a straight line represents the regression relation between brain-weight and stature better than any simple curve would. So that we are justified in concluding, as in the case of age, that until we have very much larger collections of data than are at present available we can do no better than proceed on the assumption that the regression of brain-weight on stature is linear. In fact, in these samples there is no evidence that there is any tendency towards anything but a linear relation between the two variables. The deviations of the means of the arrays from the regression line are only such as would reason-

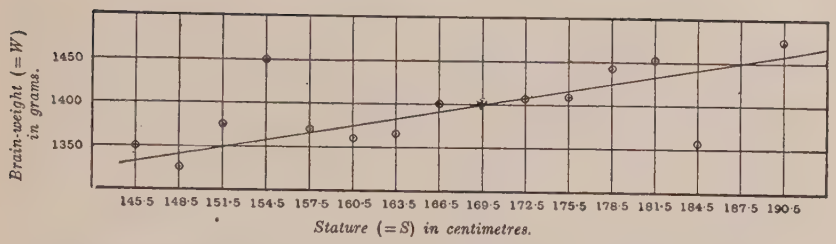


FIG. 10. PROBABLE BRAIN-WEIGHT FOR GIVEN STATURE. SWEDISH ♂ TOTAL

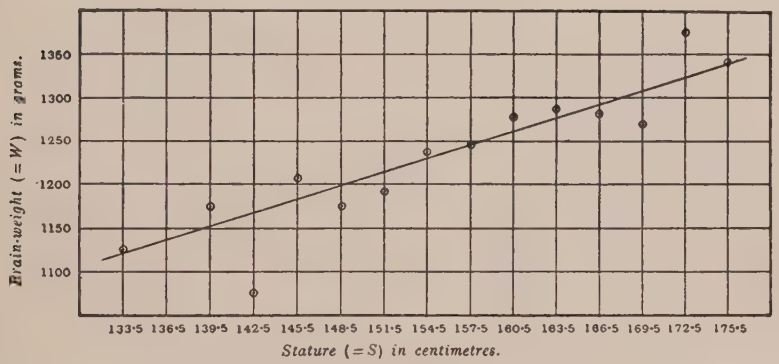


FIG. 11. PROBABLE BRAIN-WEIGHT FOR GIVEN STATURE. SWEDISH ♀ TOTAL

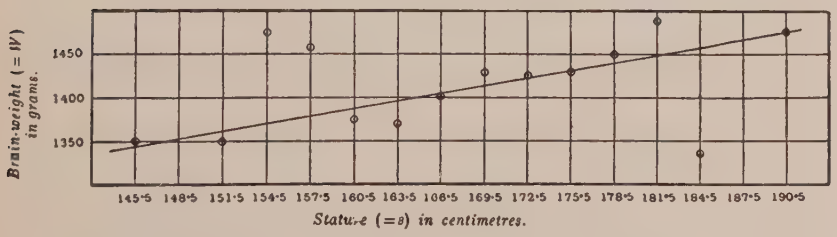


FIG. 12. PROBABLE BRAIN-WEIGHT FOR GIVEN STATURE. SWEDISH ♂ YOUNG

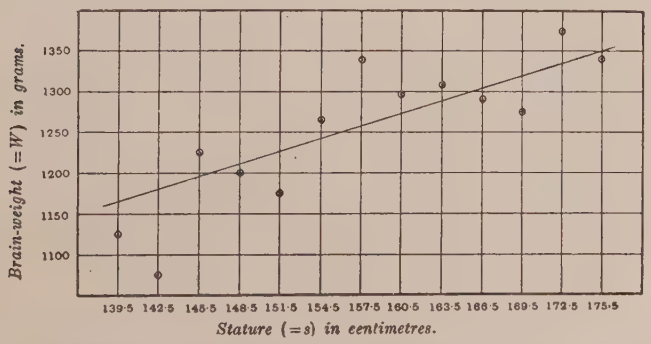


FIG. 13. PROBABLE BRAIN-WEIGHT FOR GIVEN STATURE. SWEDISH ♀ YOUNG

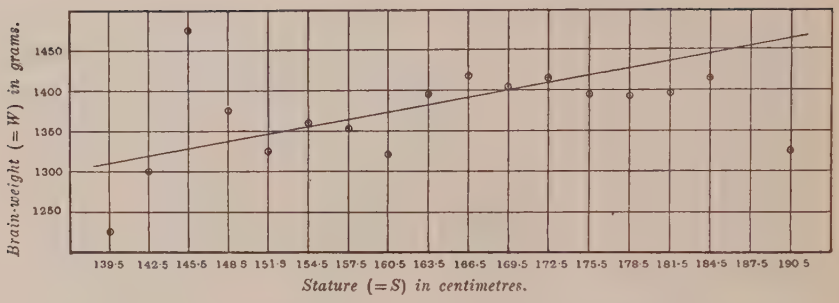


FIG. 14. PROBABLE BRAIN-WEIGHT FOR GIVEN STATURE. HESSIAN ♂ TOTAL

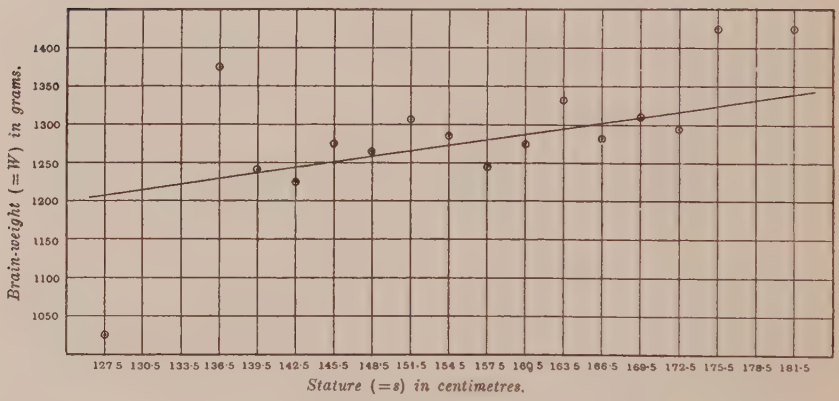


FIG. 15. PROBABLE BRAIN-WEIGHT FOR GIVEN STATURE. HESSIAN ♀ TOTAL

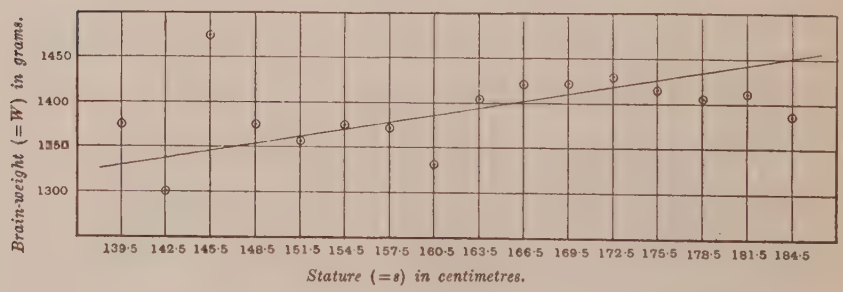


FIG. 16. PROBABLE BRAIN-WEIGHT FOR GIVEN STATURE. HESSIAN ♂ YOUNG

ably be expected to arise when we deal with relatively small samples. In connection with all of the regression diagrams it should be kept in mind that the outlying points are based on single individuals, or at most only on comparatively few individuals. Hence deviations of these outlying points from the regression line have very little or no significance as regards the general trend of the results. In fact it would really be better if the outlying points were left out entirely in the graphical representations. The non-biometrical reader should understand that each plotted point carries weight in proportion to the fraction which the number of individuals in the array on which it is based is of the total number of individuals. With this caution in mind the substantial linearity of the regression through

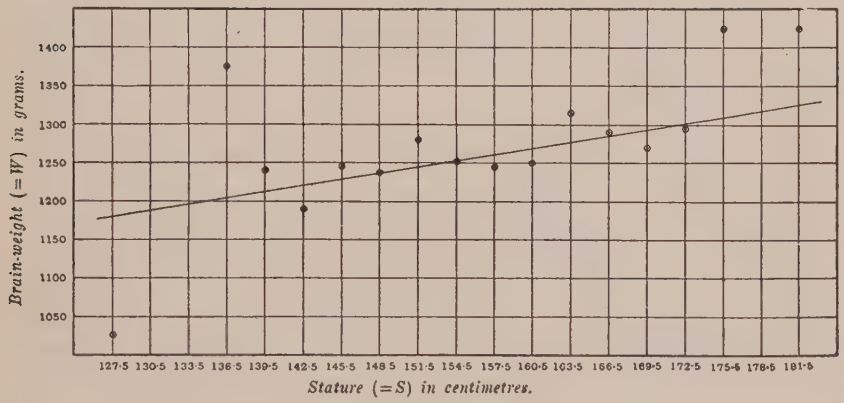


FIG. 17. PROBABLE BRAIN-WEIGHT FOR GIVEN STATURE. HESSIAN ♀ YOUNG

the bulk of the observations, which alone are of importance, becomes evident.

The regression equations showing numerically the relation between brain-weight and stature, may now be given. In these equations W denotes the probable brain-weight of an array of individuals of mean stature S or s in centimetres. As before, the equations involving S are based on the "total" series, and those involving s on the "young" series. Σ is the standard deviation of the array having the probable mean brain-weight W.

(9) Swedish ♂ $W = 915.054 + 2.859\ S$, $\Sigma = 104.533$
(10) Swedish ♂ $W = 919.374 + 2.914\ s$, $\Sigma = 107.405$
(11) Swedish ♀ $W = 421.994 + 5.234\ S$, $\Sigma = 94.421$
(12) Swedish ♀ $W = 451.643 + 5.121\ s$, $\Sigma = 99.294$

(13) Hessian ♂ $W = 913.592 + 2.857\,S$, $\Sigma = 110.787$
(14) Hessian ♂ $W = 950.214 + 2.723\,s$, $\Sigma = 109.628$
(15) Hessian ♀ $W = 834.624 + 2.714\,S$, $\Sigma = 100.643$
(16) Hessian ♀ $W = 894.031 + 2.460\,s$, $\Sigma = 100.161$
(17) Bavarian ♂ $W = 836.667 + 3.127\,S$, $\Sigma = 118.452$
(18) Bavarian ♀ $W = 627.157 + 3.824\,S$, $\Sigma = 103.562$

We may now proceed to the general conclusions to be drawn from the data given above regarding the relation of brain-weight to stature. In the first place it is to be noted that the correlation coefficients between these two variables are for each series of the same general order of magnitude as those between brain-weight and age, though of course positive instead of negative, as they are in that case. Or it may be concluded that during adult life, at least, brain-weight is only a very little more closely related to general size of body, in so far as we may take stature as a measure of this, than to age.

As in the correlation of brain-weight with age, the females have uniformly a higher degree of correlation between stature and brain-weight than the males. I am inclined to think that the same general sort of explanation may be given in this case as in that of the brain-weight and age correlations. The correlation between brain-weight and stature and the regression of brain-weight on stature are sensibly the same for the early adult period of life as for the total adult period. Without any question, I think, this correlation is to be regarded in its origin as a growth correlation, the relation between the two variables being practically fixed at the end of the growth period. After that time the relation undergoes no marked change throughout the remainder of life, since both stature and brain-weight regress on age at not far from the same rate.

Finally, the combined effect of stature and age on the weight of the brain may be examined. This relationship can best be examined through the medium of multiple regression equations of the form:

$$x_1 = \beta_{12}x_2 + \beta_{13}x_3,$$

in which β_{12} and β_{13} are the partial regression coefficients of x_1 on its associated variables x_2 and x_3, it being understood that x_1, x_2 and x_3 here stand for deviations from the means of the respective variables. For our material these equations, when reduced to absolute magnitudes, take the following forms, the significance of the letters being the same as in the regression equations given before.

(19) Swedish ♂ $W = 1091.021 + 2.288\,S - 1.755\,A$, $\Sigma = 101.819$
(20) Swedish ♂ $W = 1080.715 + 2.362\,s - 1.856\,a$, $\Sigma = 106.477$
(21) Swedish ♀ $W = 561.433 + 4.679\,S - 1.078\,A$, $\Sigma = 93.066$
(22) Swedish ♀ $W = 533.407 + 4.854\,s - 1.092\,a$, $\Sigma = 98.923$
(23) Hessian ♂ $W = 942.154 + 2.989\,S - 1.181\,A$, $\Sigma = 108.992$
(24) Hessian ♂ $W = 926.586 + 3.107\,s - 1.318\,a$, $\Sigma = 108.889$
(25) Hessian ♀ $W = 1005.607 + 2.244\,S - 2.173\,A$, $\Sigma = 94.261$
(26) Hessian ♀ $W = 974.382 + 2.345\,s - 1.829\,a$, $\Sigma = 98.883$

These equations give the probable mean brain-weight of an array of adult individuals of either sex, having any given age (in years) and mean stature (in centimeters). They are, I believe, the first multiple regression equations relating brain-weight to stature, age and sex, to be published. They are of interest from several points of view. Examples of their practical use have been given earlier in connection with the discussion of racial differences in mean brain-weight and later in connection with the sex differences. They afford a means whereby it is possible to make scientific comparisons of the mean brain-weight of different races, since by their use the probable brain-weight of a group of Swedes or Hessians having the same mean stature and age as the sample of the race with which comparison is to be made can be ascertained. We can in effect reduce both races to the same stature-age base and then examine the brain-weight differences. It would seem that these equations ought to prove very useful to anthropologists, who frequently wish to make comparisons of this kind. They are based on the two sets of brain-weighings which I believe to be on the whole the most reliable and trustworthy at present available. These equations also make it possible to determine the effect of indirect selection of stature and age on brain-weight.

As a practical example of this last use of these equations let us consider the following problem: By how much would it be necessary to modify the stature of a group of Swedish males, having a mean age of 45 years, in order that they might have the same mean brain-weight as a group of Bohemian males of the same mean age? The mean brain-weight of the Bohemian males with a mean age of 45.699 is 1454.839 grams. From equation (19) we have then

$$1454.839 = 1091.021 + 2.288\,\mathrm{S} - (1.755 \times 45.699)$$

in which S is the mean stature of the selected group of Swedes. Solving we find

$$\mathrm{S} = 194$$

or, in other words, our group of Swedes would have a *mean* height of 6 feet 4.4 inches! Now as a matter of fact the Swedish males with a mean age of 45.02 years have a mean stature of 169.79 cm. In order then to get from the adult male Swedish population a group having the same mean brain-weight as the adult male Bohemian population, by a selection of stature alone, it would be necessary to raise the mean stature of the Swedes 24.3 cm. or roughly $9\frac{1}{2}$ inches. Of course it is not for a moment to be supposed that evolution in brain-weight proceeds by the indirect selection of stature alone. Indeed when it is remembered that the mean stature of Bohemian males is only 169.4 cm. the example just cited shows how little the indirect selection of stature has to do with the matter. In the discussion of evolutionary problems, however, it is important to know just how much or how little effect each factor involved is capable of producing. It is in this direction of measuring the relative effect of different combinations of factors that regression equations such as those given above have great value.

It is apparent from equations (19) to (26) that stature and age in a measure compensate one another so far as their effect on brain-weight is concerned. It is of some interest to know in the particular cases how great an increase in stature compensates a given increase in age and *vice versa*. In order to show this I have prepared the following table.

An increase of 10 cm. in stature is compensated in its effect on brain-weight by an increase in age in the

Swedish males of 13.0 years Swedish females of 43.4 years Hessian males of 25.3 years Hessian females of 10.3 years	Total series only

X. BRAIN-WEIGHT AND BODY-WEIGHT

In only one of the four series of brain-weighings studied (the Bavarian) was body-weight tabulated in the returns. Recent workers on the subject have left this character out of their data on the general ground that it is of very doubtful significance or reliability in post-mortem returns from a general hospital population. Certainly it is true that it is influenced to a very marked degree by the conditions preceding death. Yet as Donaldson[41] has pointed out if we could get this character free of disturbing ante-mortem effects it would furnish the best measure of the general "size" of the body. Since "size" of the body is one of the things to which brain-

[41] H. H. Donaldson, *The Growth of the Brain.*

weight should first of all be related I decided to include in this study a consideration of the correlation of brain-weight with body-weight. I was fully aware of the weakness of the material and expected much worse results than those actually obtained. A glance at table 1 will recall the facts as to the distribution of the frequency of body-weight. The means are of course lower than they would be for the living population of the same race and age. This is to be expected. The variation is large but there is very good accord between males and females in this respect. Turning now to the correlations I have arranged in table 16 the values of r, η and Σ_M for the correlation between brain-weight and body-weight.

The result for the males is very satisfactory, indicating a quite reasonable approach to linearity of regression. The female series does not give so good a result, but it will be recalled that throughout the work this Bavarian female series has been found to be somewhat abnormal. The regression lines are shown in figures 18 and 19.

TABLE 16

Linearity of regression. Brain-weight on body-weight

RACE AND SERIES	r		η		Σ_M	
	♂	♀	♂	♀	♂	♀
Bavarian (Total).....	0.1671±0.0343	0.2260±0.0412	0.1845	0.3189	0.0782σ	0.2250σ

Through the great bulk of the observations the regression is evidently linear.[42] The male series comes very close to linearity.

The equations to the regression lines may now be given. In addition to the equation showing the regression of brain-weight on body-weight I have included multiple regression equations giving the probable brain-weight for given types of body-weight and stature together. The significance of the letters is as follows: W denotes the probable brain-weight of an array having a mean body-weight in kilograms W_B, or body-weight W_B and stature S. Σ is the standard deviation of the array.

(27) Bavarian ♂ $W = 1263.308 + 1.886\, W_B$, $\Sigma = 118.439$
(28) Bavarian ♀ $W = 1121.621 + 2.265\, W_B$, $\Sigma = 103.495$
(29) Bavarian ♂ $W = 917.748 + 2.234\, S + 1.355\, W_B$, $\Sigma = 117.711$
(30) Bavarian ♀ $W = 741.078 + 2.646\, S + 1.593\, W_B$, $\Sigma = 102.432$

The correlation of brain-weight with body-weight is of about the same degree as the correlation of the former with the stature, but the regression

[42] *Cf.* pp. 66, 69.

approaches more closely to linearity in the case of body-weight. As in the other cases the female shows the higher correlation.

The absolute increase of brain-weight with every increase of 10 kilos in body-weight is for the males 18.86 grams and for the females 22.65 grams. Equations (29) and (30) show that in each case stature contributes absolutely more to an increase in brain-weight than does body-weight.

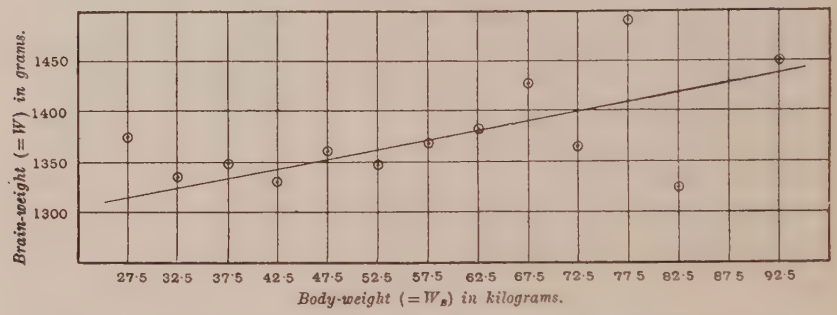


FIG. 18. PROBABLE BRAIN-WEIGHT FOR GIVEN BODY-WEIGHT. BAVARIAN ♂ TOTAL

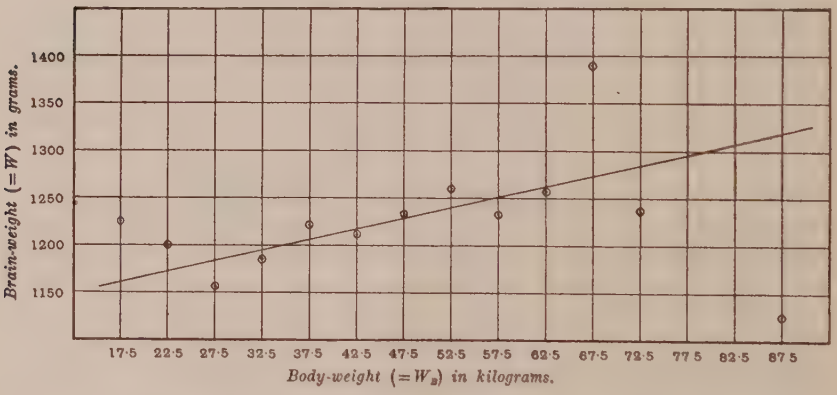


FIG. 19. PROBABLE BRAIN-WEIGHT FOR GIVEN BODY-WEIGHT. BAVARIAN ♀ TOTAL

The exact proportions are as follows: in order to bring about a change in brain-weight equal to that produced by an increase of stature of 10 cm. the body-weight would have to be increased 16.5 kilos in the males, and 16.6 kilos in the females. The greater dependence of the brain-weight on bodily characters in the female as compared with the male is very well shown in equations (29) and (30).

XI. BRAIN-WEIGHT AND SKULL CHARACTERS

As was to be expected the correlation of brain-weight with the skull length and breadth gives the highest values for the coefficients of any of the correlations dealt with. In fact one would predict on general grounds, I think, a somewhat closer degree of correlation between these characters than is actually found in the Bohemian statistics. It is likely, however, that the values here obtained (table 17) are lower than they should be on account of the methods used in measuring skull length and breadth. As has been fully described above these measurements were taken on the horizontal cut surfaces after the dome of the skull had been removed by sawing around it. This is clearly not a very precise way in which to get skull measurements. In order to show so far as may be what has been the effect of this procedure I have prepared the following tables comparing the various constants of the Bohemian skull length and breadth series with other well-known cranial series. Taking first the means we have:

RACE	SKULL LENGTH		SKULL BREADTH	
	♂	♀	♂	♀
Bohemians	176.547	170.142	149.841	144.953
Whitechapel English*	189.06	180.36	140.67	134.68
Altbayerisch†	180.58	173.45	150.47	143.98
Würtemburger†	179.48	172.74	147.88	142.90

* Macdonell, *Biometrika* vol. iii, pp. 208, 209.

† Alice Lee, *Phil. Trans.*, vol. 196, A, pp. 225–264.

The means give reasonably accordant values considering that the Bohemians are a markedly brachycephalic race. Turning next to the coefficients of variation we have:

RACE	SKULL LENGTH		SKULL BREADTH	
	♂	♀	♂	♀
Bohemians	4.173	3.233	4.441	3.815
Whitechapel English*	3.31	3.45	3.75	3.54
Bavarian†	3.37	3.57	3.89	3.39
French‡	3.97	3.65	4.21	3 67
Naqada§	3.17	3 14	3.29	3.45
Aino†	3.20	3.08	2.76	2.68

* Macdonell, *loc. cit.*, p. 220.

† Lee, *loc. cit.*

‡ Unpublished reduction of measurements in Broca's MS. by C. D. Fawcett.

§ Miss Fawcett, *Biometrika*, vol. i, pp. 438 and 456.

The Bohemian females are fairly accordant in variability with the other series in both length and breadth, but the males are apparently somewhat more variable. This greater variability in the Bohemian male series is due I believe not to less homogeneous material but to the manner in which the measurements are made. The coefficients of correlation between length and breadth may next be considered.

RACE	♂	♀
Bohemians	0.4006	0.1954
Whitechapel English*	0.240	0.350
French†	0.089	−0.042
German‡	0.286	0.488
English Criminals§	0.402	
English Middle Classes§	0.345	
Aino†	0.432	0.376
Naqada†	0.344	0.143

* Macdonell, *loc. cit.*, p. 233.

† C. D. Fawcett, *loc. cit.*

‡ Lee, *loc. cit.*

§ Macdonell, Measurements on living head and not skull. *Biometrika*, vol. i, pp. 181 and 188.

The results here are somewhat chaotic, but clearly the difference in degree of correlation between Bohemian males and females is too great to be entirely normal. It is probable that the male coefficient is somewhat too high and the female too low. The agreement between the Bohemian male coefficient and Macdonell's value for measurements on the heads of living English criminals is striking. We should expect measurements made as the Bohemian were, to agree better with measurements on the living head than with those from the skull.

The practical result to which we come is that conclusions from the Bohemian data as to the correlation between brain-weight and skull length and breadth must be only tentative until they can be tested by other less objectionable material.

We may now examine in more detail the results as to the correlation of brain-weight with these skull characters. In table 17 are given the values of r, η, and Σ_M.

The four regression diagrams concerned are figures 20 to 23.

Through the bulk of the observations the regression is very evidently linear. Figure 21 shows that the considerable deviations of η from r and Σ_M from zero in the case of the male brain-weight and skull breadth regres-

TABLE 17

Linearity of regression. Brain-weight and skull characters

RACE AND SERIES	r		η		Σ_M	
	♂	♀	♂	♀	♂	♀
Bohemians:						
Skull Length.....	0.5482±0.0273	0.3604±0.0465	0.5660	0.4032	$0.140^8\sigma$	0.1808σ
Skull Breadth.....	0.4655±0.0306	0.5041±0.0399	0.5498	0.5368	0.2926σ	0.1845σ

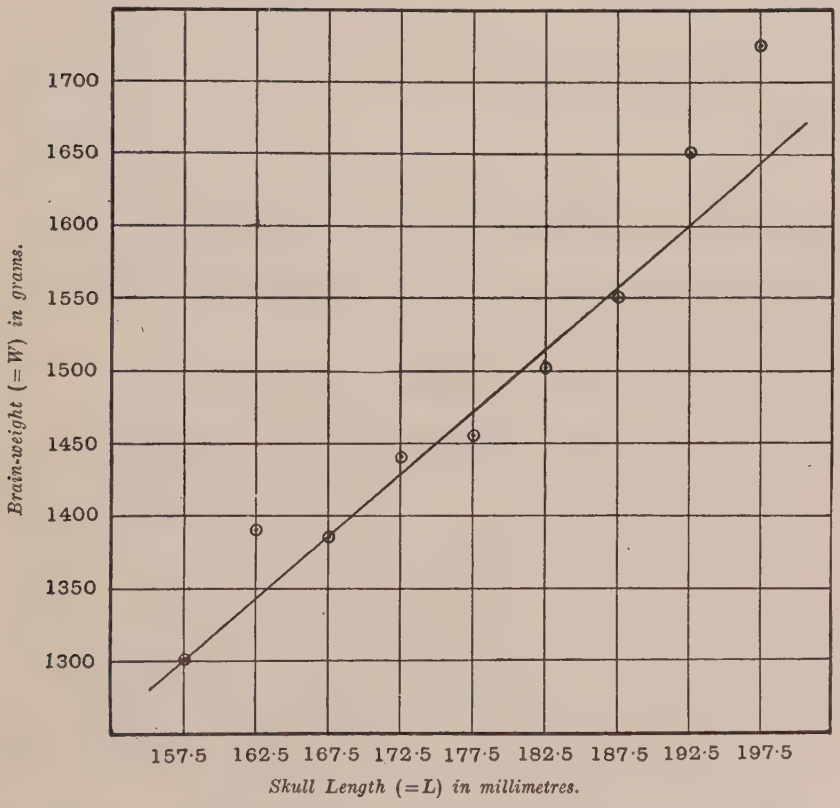


FIG. 20. PROBABLE BRAIN-WEIGHT FOR GIVEN SKULL LENGTH. BOHEMIAN ♂ YOUNG 20 TO 59 YEARS

sion are really due to the inclusion in the material of five individuals which are to be regarded as either erroneously measured or recorded, or else abnormal or extremely rare normal cases.

The regression equations follow. In these equations W denotes the probable brain-weight in grams of an array of individuals having a mean skull length L and skull breadth B in millimetres.

(31) Bohemian ♂ $W = 8.589\,L - 52.650$, $\Sigma = 96.548$
(32) Bohemian ♂ $W = 8.076\,B + 253.596$, $\Sigma = 102.170$
(33) Bohemian ♀ $W = 6.215\,L + 264.265$, $\Sigma = 88.466$
(34) Bohemian ♀ $W = 8.646\,B + 68.434$, $\Sigma = 81.908$
(35) Bohemian ♂ $W = 6.751\,L + 5.082\,B - 489.649$, $\Sigma = 99.745$
(36) Bohemian ♀ $W = 4.696\,L + 7.766\,B - 602.994$, $\Sigma = 77.893$

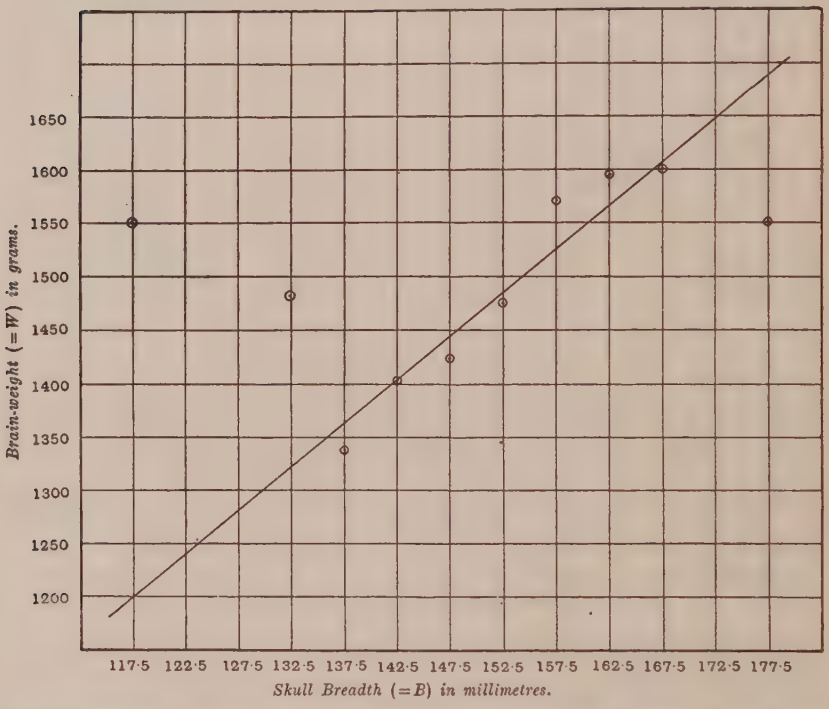


FIG. 21. PROBABLE BRAIN-WEIGHT FOR GIVEN SKULL BREADTH. BOHEMIAN ♂ YOUNG 20 TO 59 YEARS

One rather curious point may be noted here in passing. As the figures stand a smaller probable error will be made in estimating the mean brain-weight of males from a knowledge of skull length alone (equation 31) than if both length and breadth of skull are used. This apparent paradox arises for the following reason.[43] The standard deviation of the array in the regression of x_1 on its associated variables x_2 and x_3 is given by

$$\Sigma = \sigma_1 \sqrt{1 - R_1^2},$$

[43] *Cf* Yule, *loc cit.*

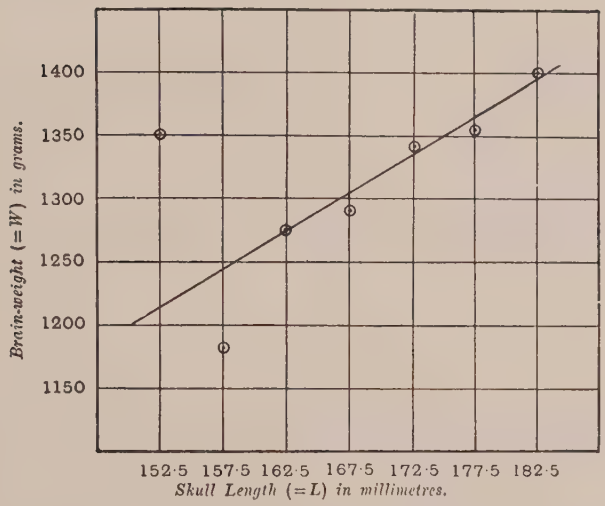


FIG. 22. PROBABLE BRAIN-WEIGHT FOR GIVEN SKULL LENGTH. BOHEMIAN ♀ YOUNG 20 TO 59 YEARS

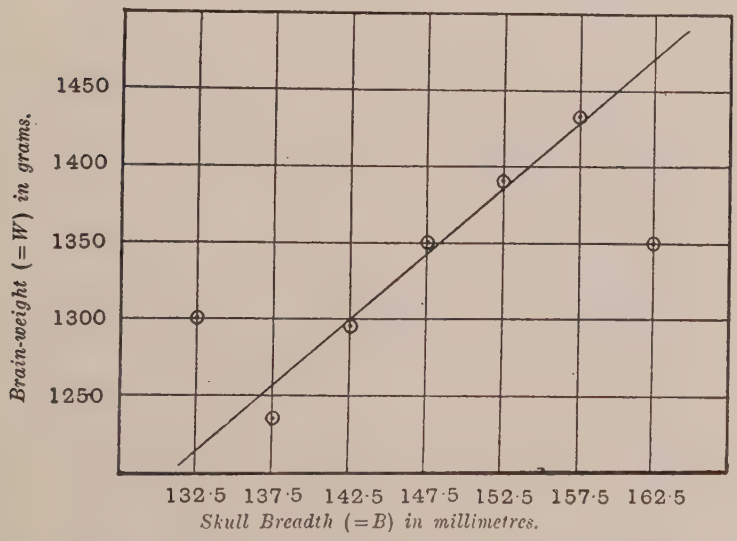


FIG. 23. PROBABLE BRAIN-WEIGHT FOR GIVEN SKULL BREADTH. BOHEMIAN ♀ YOUNG 20 TO 59 YEARS

where $$R_1^2 = \frac{r_{12}^2 + r_{13}^2 - 2r_{12}r_{13}r_{23}}{1 - r_{23}^2}$$

When the regression is of x_1 on x_2 alone the standard deviation of the array is

$$\Sigma = \sigma_1 \sqrt{1 - r_{12}^2}.$$

Now the condition under which we shall make a smaller standard error in estimating x_1 from the two variables x_2 and x_3 than in estimating it from x_2 only, is evidently that

$$(1 - R_1^2) < (1 - r_{12}^2)$$

or that $$R_1^2 > r_{12}^2.$$

Now in the case noted above $R_1^2 < r_{12}^2$, the figures being $0.2534 < 0.3005$.

Returning to the main line of the discussion, the results as to the correlation of brain-weight with skull characters are not altogether satisfactory, as they are apparently not entirely in accord with themselves. One of the points here on which information is most desired is whether brain-weight is more closely correlated with length or breadth of skull. According to our results brain-weight is more closely correlated with length of skull in the male and breadth of skull in the female. This seems a somewhat anomalous result as there is no apparent reason why one should not expect the sexes to show the same relation in the matter. However, Macdonell's (*loc. cit.*) figures for the correlation of skull capacity with length and breadth show a similar relation but in the opposite direction. He finds for the Whitechapel English skulls that the correlation between capacity and length is lower than the correlation between capacity and breadth in the males, and higher in the females. Other skull series do not, however, show this reversed relationship between the sexes. With the data at present available one cannot reach any general conclusion as to the relative closeness of correlation between brain-weight and skull length and breadth. It is very much to be hoped that future collectors of brain-weight statistics will include in their returns such head measurements as length, breadth, height and circumference taken before the cranial cavity is opened, and by standard anthropometrical methods.

A comparison of the values of the coefficients of correlation between brain-weight and skull length and breadth and between skull capacity and these characters is of interest. I have adapted the following table from Macdonell's[44] table 13.

[44] Macdonell, *loc. cit.*, p. 235.

RACE	BRAIN-WEIGHT AND SKULL LENGTH		BRAIN-WEIGHT AND SKULL BREADTH	
	♂	♀	♂	♀
Bohemians	0.5482±0.0273	0.3604±0.0465	0.4655±0.0306	0.5041±0.0399
	SKULL CAPACITY AND LENGTH		SKULL CAPACITY AND BREADTH	
English*	0.597±0.051	0.691±0.040	0.631±0.048	0.646±0.044
Naqada†	0.501±0.054	0.599±0.039	0.434±0.058	0.532±0.044
German‡	0.515±0.050	0.687±0.037	0.672±0.037	0.706±0.034
Aino‡	0.893±0.016	0.663±0.053	0.561±0.053	0.502±0.070

* Macdonell, *loc. cit.*, p. 235.
† Fawcett, *loc. cit.*
‡ Lee, *loc. cit.*

The brain-weight correlations with a few exceptions are smaller than the corresponding skull capacity correlations. A portion at least of this difference is probably to be accounted for on the ground that brain-weight fluctuates as a result of causes which do not at all affect skull capacity. Possibly the whole of the difference is to be accounted for in this way, but until there is available a more reliable collection of material on the brain-weight side the question cannot be definitely settled. Until such material is available we shall be justified, I think, in assuming that the values of the coefficients for the correlation between brain-weight and skull characters given by the male series probably represent more nearly the normal relation than do those of the female series.

Making all due allowance for the shortcomings of the material the following points are clearly brought out: (*a*) Brain-weight is positively correlated with both skull length and skull breadth. (*b*) The correlations here are higher than any of the other correlations of brain-weight with bodily characters. (*c*) The regression of brain-weight on these skull characters is approximately linear. (*d*) Indirect selection of skull characters is capable of modifying brain-weight to a considerable extent.

The use of equations (31) to (36) in selection problems has been illustrated above. One other example may be given because it bears very directly on a question of perennial interest in certain quarters. The problem to which I have reference may be stated for our present purpose in this form: Does dolichocephaly imply a greater brain-weight than brachycephaly or *vice versa*?[45] Mere inspection of equations (35) and (36) shows that

[45] Evidently the real essence of the question, so far as it has what might be called "humanistic" interest, rests on the assumption of a reasonably high positive correlation between brain-weight and intellectual ability. "Brains" not brain-weights are the things. Up to this point I have carefully avoided any discussion of the validity of such an assumption as that mentioned, deeming it entirely irrelevant to the main purpose of the study. The point is discussed in the concluding section.

neither dolichocephaly nor brachycephaly will have a great advantage over the other in respect to brain-weight, but the matter can be made somewhat more *anschaulich* by taking a particular example. Consequently I have chosen the following figures. In Macdonell's tables 1 and 2 (*loc. cit.*) are given the values for cephalic index, skull-length and skull-breadth for a dolichocephalic race (the Whitechapel English series) on the one hand and a brachycephalic race (the Altbayerisch) on the other. Those values are as follows:

♂		♀	
English.....	Cephalic index = 74.34	English.....	Cephalic index = 74.73
Altbayerisch.....	Cephalic index = 83.20	Altbayerisch...	Cephalic index = 83.10
English.....	Length = 189.06	English.....	Length = 180.36
English.....	Breadth = 140.67	English.....	Breadth = 134.68
Altbayerisch.....	Length = 180.58	Altbayerisch...	Length = 173.45
Altbayerisch.....	Breadth = 150.47	Altbayerisch...	Breadth = 143.98

Now using equations (35) and (36) for males and females respectively we have for the probable mean brain-weights of arrays having the types of length and breadth of the English and Altbayerisch series respectively, the following values:

♂	♀
English B. W. = 1502	English B. W. = 1290
Altbayerisch B. W. = 1494	Altbayerisch B. W. = 1330

In the males the dolichocephalic and brachycephalic arrays have practically equal brain-weights, while in the females the brachycephalic array gives a brain-weight about 40 grams higher than the dolichocephalic. But, as has been pointed out above, the male equations probably come much nearer representing the normal relations than do the female. On the whole, then, there seems to be little or no evidence that either dolichocephaly or brachycephaly is associated with large brain-weight.

It should perhaps be noted that no significance is to be attached to the absolute values of the brain-weights calculated from the English and Altbayerisch skull types, as indicating the probable brain-weight of those races. The differences only are of significance. The measurements of these skulls were taken only for the sake of convenience; hypothetical dolichocephalic and brachycephalic skull means would have served the purpose as well. The brain-weights obtained are of course the probable

mean brain-weight of arrays of *Bohemians* having the specified skull characters. Intra-racial regression equations can of course only be used for inter-racial prediction when the regression of all characters on one another is the same in both races, which is a condition never likely to be exactly fulfilled in practice. Of the races and characters discussed in this paper the Hessians and Swedes in brain-weight, stature, and age come nearest to fulfilling this condition.

XII. THE WEIGHT OF THE CEREBRUM

Up to this point the discussion has had to do entirely with the variation and correlation of the whole brain or encephalon. In the manuscript material placed at my disposal by Professor Donaldson data were given from which it was possible to determine the variation in the weight of the cerebral hemispheres, and the correlation of this character with stature and

TABLE 18

Variation in the weight of the cerebrum. English males

Boyd-Marshall data

CHARACTER	MEAN		S. D.	C. of V.
	No.			
Weight of cerebrum...	308	1184.940±4.859 gr.	126.438±3.436 gr.	10.670±0.293
Stature.....	308	171.602±0.268 cm.	6.976±0.190 cm.	4.065±0.111
Age.....	308	49.481±0.645 yr	16.770±0.456 yr.	—

age for English males. The results obtained from this material may now be examined. It should be stated that the records of weight were in ounces, and of stature in inches in the original material. After the constants had been calculated in ounces and inches they were transferred to the metric system in order to facilitate comparison with the other parts of the investigation. The values of the constants are exhibited in table 18.

For the correlation of weight of cerebrum with age I find

$$r = -0.1412 \pm 0.0377,$$

and for the correlation of the weight of cerebrum with stature

$$r = 0.1202 \pm 0.0379.$$

These values bring out the following points. The cerebral hemispheres are markedly more variable in weight as judged by the coefficient of varia-

tion than is the entire encephalon. This greater variation may denote a really greater variability of this part of the brain, or it may be due to the variable element which enters as a result of the separating of these organs from the rest of the brain. It seems to me likely that it is in part at least real, for the following reasons: first, the cerebral hemispheres have attained relatively enormous development late in their phylogenetic history; and again from the functional standpoint the cerebrum is the most variable part of the brain.

The correlations of the weight of the cerebrum with age and stature are both somewhat lower than the corresponding values for the other series of males falling within the ages 20 to 80. The differences can hardly be considered as significant however in view of the probable errors.

For the coefficient of regression of brain-weight on age I find $b_1 = -1.065$, and on stature $b_1 = 2.178$. By comparing these with the regression coefficients for the entire encephalon on the same characters in the series previously discussed it becomes evident at once to what a great extent changes in the total brain-weight are due to changes in the weight of the cerebrum. Unfortunately it is not possible to get relative figures here on account of lack of knowledge of the regression of the total brain-weight on stature and age for the English series. Of course the weight of the cerebrum forms a large part (ca. 87 per cent for the English series) of the total brain-weight. The problem here is to determine whether the changes in the weight of the cerebrum cause less or more than their proportional part (say 87 per cent) of the changes in total brain-weight due to stature and age changes. I have tried a number of approximate methods of getting at this question with the data available, but none of them led to any result sufficiently well grounded to warrant taking space for its publication. Putting all the results so obtained together they seemed to indicate the following conclusion, which is of course subject to modification when further material is available: viz., that changes in stature affect all parts of the brain equally (*i.e.*, in the proportion of their absolute masses) while changes in age have a relatively greater effect on the cerebral hemispheres than on the remainder of the brain (cerebellum, pons, and medulla).

The final conclusions regarding variation and correlation in the weight of the cerebrum may be summed up as follows: the cerebrum is somewhat more variable in weight than is the entire encephalon, and this character is slightly less closely correlated with age and stature. The values of the variation and correlation constants are, however, as is to be expected, of the same general order of magnitude as those for the total brain-weight.

XIII. CONCLUDING REMARKS

In concluding this study I wish to call attention to what seem to me to be some of the broader aspects of the work. There are at least two general results which alone justify its being done, I think. The first and most important is that the investigation contributes reduced material to the existing collection of biometric data on man. In the nature of the case such a collection grows slowly, but every increase in it means a definite, although it may be small, step in advance in our knowledge regarding the fundamental problems of anthropology. I regard as the second most important result the fact that it may now fairly be said that the essential trustworthiness of the most important of the existing collections of brain-weight statistics has been demonstrated, and that consequently reasoning by statistical methods on the problems involved is not of necessity lacking in validity. The agreement in the statistical constants from four series of data so divergent in their origin as those treated in this paper cannot reasonably be held to be fortuitous. It can mean, I think, but one thing, namely, that the same kind of general lawfulness underlies the variation and correlation of brain-weight and the variation and correlation in other characters of the organism. With this conclusion presumably no student of brain-weight would disagree, but some of the most eminent students have disagreed with the converse proposition that it is possible to gain a knowledge of the nature of this lawfulness by statistical methods applied to large masses of material when brain-weight is the thing concerned. This sceptical attitude owes its origin, I think, to the nature of the statistical methods which have hitherto been applied to the problem. Brain-weight statisticians have erred in two directions in the handling of their material. On the one hand, following the much-to-be-condemned practice in other fields of anthropology, entirely heterogeneous material has been grouped together to attain large total numbers. The culminating example here is, perhaps, Topinard's series of the brain-weights of 13,000 Europeans. Secondly, certain brain-weight workers have attempted to apply to the individual results gained by somewhat doubtful statistical analysis of mass data, and *vice versa*, what is much worse, have argued that the relations found to hold in a single individual might be considered to hold for the mass. As an example here might be cited Marshall's[46] attempt to correct the brain-weights of De Morgan, Thackeray, and others for stature and age. The results detailed in this chapter will indicate to the biometrician at least how dangerous both of these methods of procedure are

[46] *Loc. cit., supra.* p. 20.

with such material as brain-weight statistics. Of course both disregard fundamental canons of general statistical theory, but the chance of grave error becomes very much greater when the character under consideration shows on the one hand definite and clearly marked types in different races, and on the other hand low correlations.[47] With the application of adequate statistical methods it is possible to reach definite and significant results regarding the weight of the brain. When we have biometric constants for a considerable number of long series of brain-weight statistics from different races we shall be able to advance our knowledge greatly with reference to the general problems of the evolution of man's brain.

While the discussion of such problems can at the present time admittedly lead to only tentative results, still I think it will be worth while to examine for a moment the bearing of our results on a single evolution problem. Progressive evolution through natural selection may be brought about in any character either by the direct selection of that particular character or by the indirect selection of other characters correlated with it. As we proceed in the analysis of the problem the proportional effect of each of

[47] In this connection Weigner's paper "Ein Beitrag zur Bedeutung des Gehirngewichts beim Menschen," *Anatomische Hefte*, Bd. 23, pp. 69–109, is decidedly open to criticism. But when one, after very briefly analyzing the weighings of 69 male and 66 female brains (*sic*) from individuals ranging in age from 1½ to 82 years, by the most superficial of statistical methods, feels justified in drawing the following sweeping conclusions *inter alia*, what can biometry offer that will be in any way effective? Weigner's three principal conclusions as to fact are (*loc. cit.* p. 108):

1. "Das Gehirngewicht steht in keinem direkten Zusammenhang mit dem Alter, und wenn auch die angegebenen Zahlen sich zu Gunsten dieses Zusammenhanges verwerten einen, verlieren dieselben an der Bedeutung, da durch dieselben nicht der Beweis geführt werden kann, wie sich Gehirngewicht bei einem und demselben Individuum in verschiedenen Altersperioden verhält;

2. Zwischen dem Gehirngewichte und der Körperlänge lässt sich keine bestimmte Proportion feststellen;

3. Die Durchschnittszahl hat bloss einen relativen Wert, und zwar deshalb, weil die das Gehirngewicht repräsentierenden Zahlen durch die Wägung eines zufällig uns gerade zur Verfügung stehenden Materials gewonnen sind und das aus demselben bestimmte arithmetische Mittel keine konstante Grösse, sondern eine frei bewegliche Reihe von Zahlen uns darstellt."

It should be said that the material on which these conclusions are based is apparently within the limits of its probable errors entirely normal, and shows an approximately linear regression of brain-weight on age during adult life. This being the case the very positive conclusions seem to take on the character of extreme dogmatism. If 1. were true, by the same token no life insurance company could fix upon a premium rate for the insurance of lives of individuals, aged 21 say, which would enable it to carry on its business justly and fairly, because no life insurance company knows when any one individual aged 21 will die. Yet life insurance companies seem to be struggling on!

these two sets of factors must be quantitatively determined. A first approximation to such a determination has been here made for brain-weight and "size of body." It has been shown that of a given change in mean brain-weight between 25 and 30 per cent can be brought about by a selection of "size of body," measuring this both by stature and weight of body. As it is fair to assume that natural selection has acted on "size of body" we thus reach some idea of how much of an effect such selection has had on the weight of the brain.

A vital point at issue is as to what are the underlying causes of brain-weight differences. We have seen that there are definite racial types in brain-weight apart from differences in the other bodily characters which could be studied in this connection. To take a concrete example, we find that groups of male and female Bohemians having the same mean length and breadth of skull have probable mean brain-weights differing by about 69 grams, the female mean being lower than the male, of course. To what is this difference to be ascribed? Evidently there are a considerable number of possible factors which may enter into the matter. In the first place these males and females would still differ in stature by a certain amount so that a part of the remaining brain-weight difference would be accounted for in this way, but probably less than 25 per cent. Selecting too the same skull size would undoubtedly reduce the gross sexual difference in stature to some extent. The mean age of the arrays is essentially the same, so that nothing is gained from this source. To account for the still remaining difference there are at least the following possible factors:

1. The specific gravity of the female brain may be lower.
2. A unit increase in skull length and breadth may be associated with a greater change in skull height in the male than in the female.
3. The walls of the female skull may be thicker than in the male.
4. The female brain may fill the skull cavity less closely than does the male. (The opposite has been stated to be the case. *Cf.* Donaldson, *loc. cit.* p. 118.)
5. The ventricles of the brain may be proportionately larger in the female than in the male brain.

With the material at present available it is impossible to determine which of these possible factors really play the important rôle. It is my purpose merely to call attention to the kind of problem that confronts one at this point in the analysis of brain-weight constants. It at least will serve to show some of the directions in which more definite information is desired.

Donaldson[48] has well said: "The search for correlation between the size and form of the brain and the degree of the intelligence has interested all who have worked on this organ, and although it might be designated as the psychologist's standpoint, it has, from the very first, been in some measure before the minds of all." This point has so far been avoided in this study for the reason that it seemed to me desirable to settle the question of the purely physical variations and correlations before making any attempt on the much more difficult matter of brain-weight and intelligence correlation. I have, however, one point to add to the discussion of this already much debated subject. It follows rather simply from the theory of multiple correlation and regression that if the correlation coefficients r_{12} and r_{13} measuring the correlation between characters x_1 and x_2, and x_1 and x_3 are known, then it is possible to determine the limits between which the value of the coefficient of correlation r_{23} between x_2 and x_3 must lie. Without going into the details of the theory involved, which is given in convenient form by Yule,[49] it may be said that since

$$(r_{23} - r_{12}r_{13})^2 \text{ is not } > 1 + r_{12}^2 r_{13}^2 - r_{12}^2 - r_{13}^2,$$

r_{23} must lie between the limits

$$r_{12}r_{13} \pm (\sqrt{1 + r_{12}^2 r_{13}^2 - r_{12}^2 - r_{13}^2}).$$

Now, very fortunately, Pearson and his co-workers[50] at University College have determined for Cambridge undergraduates the correlation between "intelligence" as denoted by place in degree examinations and certain physical characters whose correlation with brain-weight we also know. Evidently then we have the material for getting at an approximation of the limits of the correlation between brain-weight and intelligence except that the two correlations from which the limits of the third are to be determined are not from the same material. This of course introduces an element of error, but as the coefficients of correlation of brain-weight with other characters seem to be fairly constant from race to race, a very serious error will probably not be made in assuming that the values of these correlations would not differ greatly in the English from what they are for continental races or from one another in two different groups of English. Leaving for the moment this question of possible error, let us examine the

[48] *Loc. cit.*, p. 85.

[49] *Loc. cit.*

[50] Lee, A., Lewenz, M. A., and Pearson, K., On the correlation of the mental and physical characters in man, Part ii, *Roy. Soc. Proc.*, vol. lxxi, pp. 106–114, 1902.

figures, first taking this problem: Assuming that Cambridge undergraduates have the weight of the cerebrum correlated with stature to roughly the same degree as is shown in Boyd's English data, what will be the limits of the correlation between weight of cerebrum and intelligence? The necessary data are as follows:

$r_{12} = -0.0056$ correlation coefficient between stature and intelligence
$r_{13} = 0.12$ correlation coefficient between stature and weight of cerebrum

The limits of r_{23}, or of the coefficient of correlation between brain-weight and intelligence, are roughly $+0.98$ and -0.99. Now r_{12} is really insignificant, the conclusion the authors draw being that (*loc. cit.*, p. 107), "stature is not correlated with place in degree examinations." Putting $r_{12} = 0$ the limits of r_{23} are $\pm \sqrt{1 - r_{13}^2}$. Or the practical conclusion we reach is that, so far as the known correlations of weight of cerebrum and intelligence respectively with stature give evidence, the correlation between weight of cerebrum and intelligence may have any value between 0 and ± 1. There is no evidence from this source as to whether the correlation is positive or negative. Suppose we take still another case, stating the problem this time in this way: Assuming that the Cambridge undergraduates have brain-weight correlated with skull length to roughly the same degree as is shown by Bohemian males, what will be the limits of the correlation between brain-weight and intelligence? The necessary coefficients are:

$r_{12} = 0.0861$ correlation coefficient between skull length and intelligence.
$r_{13} = 0.5482$ correlation coefficient between skull length and brain-weight.

The limits of r_{23} or the correlation between brain-weight and intelligence, are $+0.7$ and -0.6, or practically we reach much the same result as before. As to the reliability of these limits this may be said: in order to fix correctly the sign of the correlation between brain-weight and intelligence, both the coefficients between intelligence and physical characters and brain-weight and the same physical characters would have to be greater than $\sqrt{0.5}$ or 0.707. Now I think it is reasonably evident from what we already know, that for civilized man at least there is no likelihood whatever of ever finding values as high as 0.707 for the coefficients concerned. Or in other words the degree of the correlation between brain-weight and intelligence is indeterminate, with the probability that it is sensibly equal to zero. That is, brain-weight and intelligence in the sense of mental capacity are probably not sensibly correlated. All the inferential evidence when subjected to careful scrutiny leads to the same conclusion, I think

In closing I may perhaps be permitted to point out what seem to me to be the most promising lines for future investigation of the problems regarding the weight of the brain. In the first instance it seems to me highly desirable to transfer the problem for a time at least from man, where in the nature of the case all the fundamental records must be vitiated from the fact that we cannot weigh the brains of any considerable number of normal men instantly killed. As a consequence of this impairment of the data resulting from complex ante-mortem conditions the *absolute* values of certain of the biometric constants concerned cannot in the case of man be proven to be sensibly identical with what we should consider necessarily normal values. To be sure, such a sensible identity cannot, on the other hand, be disproven, but a state in which one can neither prove nor disprove is scientifically not altogether satisfactory. By working with animals it is possible to weigh any number of brains of instantly killed normal individuals. From records so obtained the values of the important biometric constants like the coefficients of correlation can be satisfactorily determined. For continuing the work on the human side the primary need is for more and larger collections of brain-weights in which close attention is paid to the racial homogeneity of the material and to the recording of other subsidiary facts as well as brain-weight. In comparison with the present series of brain-weighings a series which should record the following facts would be almost ideal.

1. Brain-weight, by the standard method
2. Race. In this the more essential detail given the better
3. Country of residence during adult life
4. Occupation (as an index of social status)
5. Immediate cause of death
6. Chronic diseases of adult life
7. Stature
8. Body-weight
9. Head length
10. Head breadth
11. Head height
12. Maximum horizontal circumference of head

(9–12: Measurements all made before skull is opened and with hair removed at points of contact)

13. Age
14. Sex

Such a list at first sight appears formidable from the practical standpoint, but evidently the things which would give the greatest difficulty are 2 to 6 inclusive. The head measurements could be made with little trouble, and a relatively small expenditure of time. This list is presented with the hope that any future workers who may be about to

undertake the great labour involved in obtaining a large mass of human brain-weight statistics will at least consider the points raised. The few additional facts would take but little more time in the collection, and they would greatly enhance the value of the completed series. I would especially call attention to the need for larger series of brain-weight statistics than those we now have. In order to do really "close" statistical work on the subject it is desirable that we have larger arrays of individuals of given age and stature types in order that the regressions may be smoothed and the brain-weights in more absolutely homogeneous material may be investigated.

Finally it should be said that the present investigation is, from the biometrical standpoint, only a "first" study of the problems of brain-weight correlation. Much yet remains to be done, and as will have been apparent to the reader could be done with the present material. One thing especially which might be done is to separate the material into a third "old" group, comprising the individuals falling in age between 50 and 80, and treat this group separately. I very much doubt, however, in view of the apparent substantial linearity of the regression of brain-weight on age through the whole of adult life, and considering the statistically small number of individuals which in each case would have been available, whether the value of the results so obtained would have been in any way commensurate with the labour involved. Even a biometrician must stop somewhere. When we have much larger collections of brain-weight statistics to work with an old age division of the material will be at least experimentally justified.

XIV. SUMMARY

Some of the more important general conclusions of a study of five series of brain-weighings, representing Swedish, Hessian, Bavarian, Bohemian, and English sub-races of man and including altogether the weights of 2100 adult male and 1034 adult female brains, may be summarised as follows:

1. There are definite racial types in brain-weight. The differences between racial groups in this character are only in part to be accounted for by differences in other characters of the body.

2. The series studied exhibit a very fair degree of homogeneity.

3. In respect to variability the characters, brain-weight and skull capacity are sensibly equal. The coefficients of variation for brain-weight are intermediate in value between those which have been determined for skeletal characters in man on the one hand, and those for the weights of the viscera and for various physiological characters on the other hand.

4. Having regard to the size of the present series we conclude that variation in weight of the brain may for practical purposes be considered to

follow the "normal" law of the distribution of errors. If the mean and the mode do not exactly coincide the mean will be slightly greater than the mode in brain-weight frequency distributions.

5. The correlation of brain-weight with age, stature and body-weight, is in all cases low.

6. The correlation of brain-weight with skull length and skull breadth is, in comparison with the other characters studied, fairly high, and is for both skull length and breadth positive.

7. The sexual differences in mean brain-weight are practically constant in all the races studied, whether considered absolutely or relatively.

8. Only a part of the sexual difference is to be accounted for by differences between the sexes in other bodily characters.

9. The sexes are equally variable in respect to brain-weight.

10. The weight of the brain tends to be more highly correlated with other characters in the female than in the male.

11. The correlation of brain-weight with age is negative. The regression of brain-weight on age is, so far as can be determined from our present series, linear throughout the period of life comprised between the ages 20 to 80. In other words, there is a steady decline in the weight of the brain with advancing age, beginning at about the twentieth year and continuing throughout adult life.

12. The correlation of brain-weight with stature and with body-weight is positive and in each case the regression is linear.

13. The weight of the cerebrum follows the same laws of variation and correlation as does the weight of the entire brain, but exhibits a somewhat greater relative variability.

14. The results as to variation and correlation in brain-weight are closely accordant for all the races studied.

15. The indirect selection of bodily characters can modify the mean weight of the brain to a considerable extent. The amount of this effect has been measured in certain cases.

16. There is no evidence that brain-weight is sensibly correlated with intellectual ability. The limits of this correlation have been shown to be not closer than 0 and $\pm$ 0.6.

17. So far as can be determined from present material dolichocephaly has associated with it no advantage in mean brain-weight over brachycephaly and *vice versa*.

18. There is great need for further large and homogeneous collections of brain-weight statistics. When these are available for a considerable number of races it will be possible to pass from intra-racial to inter-racial problems.

CHAPTER II

Intelligence and the Size of the Head[1]

On account of the considerable intrinsic interest of the question as to whether or not there is a sensible degree of correlation between intellectual capacity and the size of the brain or of the head it seems desirable to have all the exact and direct evidence on the matter which can be obtained. It has been alternately maintained that such a correlation does exist, and that it does not exist, but until comparatively recently there have been no attempts to obtain by adequate methods any definite quantitative statement regarding this question. It is, I think, perfectly obvious that the only way in which a definite answer may be obtained to the problem is to apply proper methods of reduction to a sufficiently large body of homogeneous statistics which record for each individual some measure of the intellectual ability on the one hand and of the size of the brain or head on the other hand. It is idle to attempt to reach any answer by an examination of individual cases, because, to name one point only, our general experience teaches beyond question that if such a correlation exists at all it is of a low order, and in consequence even its existence, to say nothing of its amount, cannot be determined from individual instances. The important thing to determine first of all is whether, if we take a large sample out of a homogeneous population, the individuals of more than average intellectual ability possess brains or heads above the average in size. It is to get evidence on this direct question of fact that, so far as I understand it, the biometrical work on the problem has been undertaken.

The purpose of this chapter is to present the results of the reduction of some statistics published in 1905, and which show, for a reasonably homogeneous German population, the relation between the horizontal circumference of the head and intelligence. These admirable statistics were collected by Dr. G. Eyerich and Dr. L. Loewenfeld and are published in a memoir by these authors, bearing the title: "Uber die Beziehungen des Kopfumfangs zur Körperlänge und zur Geistigen Entwicklung."[2] The

[1] This paper was originally published, in practically its present form, in *Jour. Comp. Neurol. and Psychol.*, vol. 16, pp. 189–199, 1906, under the title "On the correlation between intelligence and the size of the head."

[2] Wiesbaden, J. F. Bergmann, 1905, pp. 55.

statistics which are given in most complete form in the memoir provide data for 935 Bavarian soldiers in the ordinary two-year service, regarding intelligence and head circumference. The data are recorded in two tables the first of these gives as one variable the head circumference of the individuals in 0.25 mm. classes, and as the other variable the "intellektueller Begabung," the individuals being sorted into four classes designated as follows: "I, sehr gut beanlagt"; "IIa, gut beanlagt"; "IIb, normal"; "III. beschränkt." The second table gives the same data with classes IIa, and IIb, of the intelligence scale combined into one, the rubrics then being "sehr gut," "gut," and "schwach." As the only data of a similar kind which have hitherto been analyzed biometrically are those of Pearson on Cambridge undergraduates[3] and English school children,[4] it seemed to me desirable to reduce these German data in order that comparisons might be instituted.

The all-important matter in dealing with data of this character is to know something about the basis of the classification on the intelligence side. If there is any constant bias in the distribution of the individuals into whatever "intelligence" classes are adopted it is clear that the resulting statistics will lose all significance so far as our problem is concerned. In order that as clear an idea as possible may be gained of the nature of this German material in this respect it seems best to quote in full what the authors say in regard to the matter. On page 26 of the memoir referred to is the following statement:

Um die Verwertung des gesammelten Materials zu vereinfachen, haben wir die untersuchten Personen bezüglich ihrer geistigen Befähigung in 3 Klassen gesondert: in solche von mittlerer, i.e., durchschnittlicher Begabung und in solche, deren Begabung über und unter dem Durchschnitt steht. Die uns vorliegenden Angaben über die geistige Qualifikation der untersuchten Leute ermöglichten jedoch die Trennung der Durchschnittsgruppe in 2 Untergruppen und lassen auch eine solche wünschenswert erscheinen: eine Gruppe, die den über dem Durchschnitt stehenden sich nähert, und eine solche, die mehr an die unter dem Durchschnitte heranrückt. Es entgeht uns hierbei keineswegs, dass die Beobachtungen, auf welche sich unsere Qualifikationen stützen mussten, z.T. unzulänglich und nicht ganz einwandfrei sein mögen. Es konnte nämlich im Allgemeinen neben der auf das dienstliche Verhalten sich beziehenden Qualifikation seitens der militärischen Vorgesetzten zunächst nur ein [illegible] den einzelnen Untersuchten geliefertes Elaborat über ihren Lebensgang bis zum Eintritt in die Armee verwertet werden. Das militärische Leben ist begreiflicherweise nicht geeignet, einem Individuum Gelegenheit zur Entfaltung aller seiner geistigen Gaben zu geben, und so mag wohl ein mit gewissen,

[3] Pearson, K., On the correlation of intellectual ability with the size and shape of the head, *Proc. Roy. Soc.*, vol. 69, pp. 333–342.

[4] Lee, A., Lewenz, M. A., and Pearson, K., On the correlation of the mental and physical characters in man, part II, *Proc. Roy. Soc.*, vol. 71, pp. 106–114.

z. B. künstlerischen Talenten Ausgestatteter, militärisch eine schlechte Zensur erhalten. Die Verhältnisse liegen jedoch derart, dass wir für die Beurteilung des allgemeinen Standes der Intelligenz, auf den es uns in erster Linie ankommt,—einseitige Talente kommen sogar bei Schwachsinnigen vor—die militärische Qualifikation als wohl verwertbar ansehen müssen. Die Qualifikation erfolgt nämlich erst nach längerer Beobachtung der in den Dienst eingestellten Individuen. Ein Mensch von mittlerer oder über dem Durchschnitt stehender intellektueller Begabung mag sich bei den dienstlichen Übungen ungeschickt benehmen, es auch an der nötigen Aufmerksamkeit fehlen lassen. Er wird aber dann beim Unterricht seine Fähigkeiten zur Geltung bringen und in seinem Curriculum vitae ebenfalls einen Index für den Stand seiner Intelligenz liefern. Erhebliche Irrtümer bezüglich der geistigen Klassifizierung der Untersuchten, etwa derart, dass ein Beschränkter als wohlbegabt und umgekehrt angesetzt wurde, scheinen uns daher ausgeschlossen. Wir haben übrigens in einzelnen Fällen, die uns spezieller Aufklärung bedürftig erschienen, es nicht an eingehenderen Nachforschungen über das geistige Verhalten der Betreffenden fehlen lassen.

From this statement I think we may fairly conclude that the statistics were properly collected, and probably give a fair representation of the distribution of this population with respect to general intellectual ability. That there was no significant, constant bias working in the collection of this material seems to me also very probable from the following fact: The conclusion which Drs. Eyerich and Loewenfeld reach is that there is no definite relationship between intelligence and the size of the head. Hence it seems reasonable to suppose that any subconscious tendency which might have existed toward biasing the returns would have been likely to be operative in the direction of making the statistics show no correlation between these variables. Yet as we shall see later the statistics when subjected to analysis show a slight but very regular and perfectly definite tendency toward the association of more than average intelligence with more than average head circumference. The statistics are, I believe, on the whole trustworthy.

The raw material is given in detail in tables 19 and 20, which are copies of tables IV and V of the original memoir. In table 20 the observed frequencies are given in italic type: the ordinary figures in each compartment of the table give the proportionate frequency which should fall in that compartment on the basis of independent probability (*cf. infra.*).

In dealing with this material I first applied the contingency method[5] to table 20 as it stands. It was apparent as soon as the independent probability of occurrences for each compartment of the table had been calculated that there were very considerable and regular deviations of the ob-

[5] Pearson, K., Mathematical contributions to the theory of evolution. XIII. On the theory of contingency and its relation to association and normal correlation. *Drapers' Company Research Memoirs, Biometric Series,* I, pp. 1–35, 2 pl., 1904.

TABLE 19

FÄLLE	KOPFUMFANG	I. SEHR GUT BEANLAGT	IIA. GUT BEANLAGT	IIB. NORMAL	III. BESCHRÄNKT
1	50.50				1
1	50.75		1		
1	51.75	1			
2	52.00			1	1
1	52.25				1
1	52.50		1		
1	52.75				1
5	53.00		1	1	3
7	53.25		1	4	2
12	53.50	2	3	5	2
6	53.75	1	3	1	1
22	54.00	3	5	6	8
21	54.25	2	7	5	7
29	54.50	6	6	11	6
29	54.75	6	11	10	2
55	55.00	11	16	17	11
43	55.25	4	15	15	9
45	55.50	6	13	15	11
36	55.75	6	10	11	9
107	56.00	19	33	40	15
74	56.25	16	18	22	18
57	56.50	13	15	21	8
44	56.75	6	15	18	5
92	57.00	22	31	29	10
40	57.25	8	13	12	7
44	57.50	6	13	16	9
28	57.75	4	7	15	2
49	58.00	12	17	15	5
27	58.25	4	11	8	4
16	58.50	5	3	6	2
9	58.75	1	3	3	2
15	59.00	4	5	5	1
4	59.25	2	2		
6	59.50	1	1	4	
1	59.75		1		
2	60.25		1		1
1	60.50			1	
1	61.00	1			
935					

servations from what would be expected if intellectual capacity and head circumference were in no way associated in this sample. If these two characters were entirely independent we should expect the frequency

in each compartment of table 20 to be, within the limits of error due to random sampling, the same as that given by the ordinary figures. But it is clear that the italic figures and the ordinary ones differ from each other in a definite way and by considerable amounts. Thus, neglecting the arrays of low frequency as too small to be significant, we see that in the left hand column (intelligence above average) individuals with small

TABLE 20

KOPFUMFANG	FÄLLE	KLASSIFIKATION		
		Sehr gut	Gut	Schwach
50–51	*2*		*1*	*1*
		0.37	1.28	0.35
51–52	*1*	*1*		
		0.18	0.64	0.18
52–53	*5*		*2*	*3*
		0.92	3.20	0.88
53–54	*30*	*3*	*19*	*8*
		5.52	19.21	5.26
54–55	*101*	*17*	*61*	*23*
		18.58	64.70	17.72
55–56	*179*	*27*	*112*	*40*
		32.93	114.67	31.40
56–57	*282*	*54*	*182*	*46*
		51.87	180.65	49.46
57–58	*204*	*40*	*136*	*28*
		37.53	130.68	35.78
58–59	*101*	*22*	*66*	*13*
		18.58	64.70	17.72
59–60	*26*	*7*	*18*	*1*
		4.78	16.66	4.56
60–61	*3*		*2*	*1*
		0.55	1.92	0.53
61–62	*1*	*1*		
		0.18	0.64	0.18
	935			

heads occur in defect of the expected proportions, and those with large heads occur in excess of expectation. The reversed relation holds for the right hand column (intelligence below average).

From table 20 as it stands I find the value of the mean square contingency (*cf.* Pearson, *loc. cit.*) to be:

$$\Phi^2 = 0.0385$$

which leads to a value of the contingency coefficient of

$$C_1 = 0.1925$$

Using the method of mean contingency

$$\Psi = 0.0397$$

whence $C_2 = 0.13$

Now, if the variation follows the normal law and the grouping is not too fine, the value of C calculated by the two methods should be equal. This is clearly not the case. Hence, it is necessary to determine whether, by taking a somewhat coarser grouping, these values may not be brought nearer together.[6] Accordingly, a table was formed in which the size of the head circumference classes was doubled, thus reducing the number of rows from twelve to six. This new table then had eighteen compartments with some observed frequency in each. Working from this table I found the following values:

$\Phi^2 = 0.0203$
$C_1 = 0.1410$ (mean square contingency coefficient)
$\Psi = 0.0426$
$C_2 = 0.14$ (mean contingency coefficient)

These values are in very good accord and we may hence conclude that with this grouping

$$C_1 = C_2 = r = 0.14$$

It has been shown by Pearson that it is likely that the probable error of a contingency coefficient will be less than

$$2 \times 0.67449 \frac{1-C^2}{\sqrt{N}}$$

For $C = 0.1410$ and $N = 935$ the value of the above expression is ± 0.0432. Assuming that this in any event does not exceed the true value of the probable error we conclude that the data in table 20 exhibit a positive correlation between general intelligence and head circumference expressed by a coefficient of

$$r = 0.14 \pm 0.04$$

[6] A full discussion of the effect of too fine grouping on the value of the contingency coefficient is given in the original memoir on the contingency method, to which the reader is referred.

In other words, the chance that such a system of frequencies as that given in table 20 would arise if, in the population under consideration, there were no correlation between head size and intelligence would be, so far as we can judge, not greater than 1.82 in 100. It seems reasonable to conclude, therefore, that this material does probably show a sensible correlation between these characters.

We may, however, test the matter further. From the data furnished by table 19 it is possible to form a fourfold table, and evaluate the coefficient of correlation directly and with a known probable error by the method given by Pearson.[7] Such a fourfold table is given as table 21.

The division for head circumference was taken as nearly as possible at the mean.[8] From this table I find the following values for the constants:

$$h = 0.03620, \qquad k = 0.08456,$$

whence

$$0.000635\,\Theta^4 - 0.001409\,\Theta^3 - 0.001531\,\Theta^2 + \Theta - 0.103222 = 0$$

Accordingly

$$r = 0.103 \pm 0.034.$$

[7] *Phil. Trans.*, vol. 195 A, pp. 1–46.

[8] The constants for the head circumference distribution as given in table 20 were found to be as follows:

Mean = 56.499 ± 0.031 cm.
Standard deviation = 1.413 ± 0.022 cm.
Coefficient of variation = 2.501 ± 0.040 per cent

These values are in close accord with those obtained from well-known homogeneous series, and point clearly to the very substantial *physical* homogeneity of the present sample. Indeed this fact is so striking that I cannot refrain from reproducing for comparison the following tables of coefficients of variation for horizontal circumference from Macdonell's memoir on the skull (Biometrika, vol. 3, p. 223). I insert in the table the coefficient deduced from the present work.

AUTHORITY	RACE	HORIZONTAL CIRCUMFERENCE	
		♂	♀
Macdonell	English	2.87	2.92
Pearson	Bavarians	2.86	3.09
Pearson	Modern Badensians	3.02	2.34
Pearson	Row Grave Germans	2.70	2.40
Fawcett	Naqada	2.54	2.27
Eyerich and Loewenfeld (Pearl)	Bavarians (living head)	2.50	

Actually the present data show the least variability of any of the male series in spite of the fact that the measurements were made with a tape on the living subjects.

Or, the coefficient of correlation is almost exactly three times its probable error. The chance, then, of such a system of frequencies as that of table 21 arising if intelligence and head circumference were not correlated in the population from which the sample is drawn is approximately 4.30 in 100. We may reasonably conclude then that there is probably a small correlation between these characters.

There is one further point needing discussion. It will be noted that the coefficient of contingency deduced from table 20 is somewhat greater than the coefficient of correlation from table 21 while at first thought it might be supposed that according to the theory of contingency they ought to be equal. This, however, does not follow, because the division of the intelligence scale in tables 19 and 21 is different from what it is in table 20 on which the contingency coefficient is based. In tables 19 and 21 the middle class of table 20 is divided. Now, I think it will be admitted that

TABLE 21

Correlation between intelligence and head circumference

	INTELLIGENCE		
HEAD CIRCUMFERENCE	Below average	Above average	Total
Below 56.50	272	227	499
56.50 and above	209	227	436
Totals	481	454	935

there would be far more doubt in the mind of the classifier as to whether a given individual ought to be placed in class IIa or IIb of table 19, than as to whether he should be placed in the "gut" class of table 20. But any errors made in the assorting of individuals into the two middle classes of table 19, will affect the coefficient of correlation deduced from the fourfold table, while, of course, they would in no way affect the contingency coefficient. Consequently I am inclined to think that in this case the contingency coefficient is a truer measure of the real degree of correlation. In any event, the difference between the contingency coefficient and the correlation coefficient from the fourfold table is only of the order of the probable error of the latter.

To sum up, then, we find by analyzing fairly copious statistics forming a homogeneous sample of the males in the poorer classes of the Bavarian population that there is a low, but still probably sensible, positive correlation between the horizontal circumference of the head and general intelli-

gence. This result appears to be of considerable interest. The only previous statistics of a similar nature are Pearson's data[9] for Cambridge undergraduates, and English school children.

In order to show how these results compare with those of the present discussion I have taken the mean of the nine coefficients for the correlation of absolute head dimensions (length, breadth and auricular height) with intelligence which Pearson has given. The value is 0.0736. All of the nine coefficients are positive.[10] To these values we are now able to add the coefficient for the correlation between intelligence and another head measurement, *viz.*, horizontal circumference, with a value of 0.14 $\pm$ 0.04. While some of the coefficients given by Pearson are, when taken by themselves, insignificant in comparison with their probable errors, we must give due weight to the fact that the sign of the correlation when we deal with absolute head measurements and intelligence is in all cases positive.

In general I think the reasonable conclusion to draw is that from the data available it seems fairly probable that there is a sensible, but *very slight*, positive correlation between intelligence and size of head. It will be understood that any conclusion regarding this matter must for the present be more or less tentative. It is perfectly clear that we are dealing here with a correlation of a very low order, the *general* existence of which cannot be definitely asserted till we have further statistics covering a wide range of social classes of different races. How slight this correlation must in general be is indicated by the fact which has been brought out in the preceding chapter[11] that what are probably the best series of brain-weight statistics available give no definite evidence of the existence of a positive correlation between that character and intelligence. The results from the statistics analyzed in the present chapter, though the material was drawn from a very different population, essentially confirm Pearson's conclusion that "there is no *marked* correlation between intelligence and the size or shape of head."

Suppose it be granted that it is a fact that there is, however, a slight, though sensible correlation between size of head and intelligence, what interpretation

[9] *Loc. cit.*

[10] I am informed by Professor Pearson that since the preliminary papers here cited were published, the material on which they were based has been worked over anew by the contingency method. The result has been to give slightly higher values to some of the coefficients and a generally smoother system. These new values are thus in even better accord with the coefficient found in the present work for intelligence and horizontal circumference.

[11] See the preceding chapter of this book.

are we to put upon the fact? It seems to me that it would be absolutely fallacious to base upon these data any general argument that "men of genius have large heads." I quite agree with Professor Pearson in his conclusion that: "For practical purposes it seems impossible, either in the case of exceptionally able men or in the bulk of the population, to pass any judgment from size of head to ability or *vice versa*." If further statistics (of which there is great need) should show that generally there is a just sensible positive correlation between these characters, the correct interpretation of the fact would, it seems to me, probably be physiologic rather than psychologic. That is to say, the association between vigor in growth processes (leading to a well-developed body) and vigor in mental processes would most probably be the result of the action of good conditions of nurture. Other things being equal groups of men with well-nourished bodies are on the average likely to be more able intellectually than groups in which bad conditions of nutrition prevail. Such an interpretation of the facts seems at present to have much better justification than any which in effect implies that a big brain connotes *per se* an able mind.

Rightly interpreted the facts regarding the correlation between size of head and intelligence seem to me simply to furnish, so far as they go, direct statistical evidence in favor of the adage: *Mens sana in corpore sano.*

CHAPTER III

Race Crossing and the Sex Ratio[1]

There would appear to be widely prevalent among practical stock breeders an opinion that the relative proportion of the sexes may be influenced by the method of breeding practiced. As evidence of the existence of such an opinion two citations will suffice. Others might be given. Davenport in his memoir on *Inheritance in Poultry*,[2] introduces a section on "Sex in Hybrids" (p. 97) with the statement that: "There is a widely held and frequently expressed opinion that hybrids show an excessive proportion of males." He further says that: "Bateson and Saunders probably have this in mind in their statement—'the statistical distribution of sex among first crosses shows great departure from the normal proportions.'" No support is given to the view that hybrids show an undue proportion of males by Davenport's own statistics, the general conclusion being that: "The exceptions to the law of equality of sexes in hybrid offspring are . . . . individual and not of general significance."

It is a matter of interest to note that while the opinion appears to be widespread that the kind of breeding practiced influences the sex ratio there is not entire uniformity as to what the influence of a particular method of breeding on sex is. Thus one would infer from a statement in a recent work by Müller[3] that it has been generally held by continental breeders, at least, that *inbreeding* tends toward the production of an unduly large proportion of males. Müller[3] (*loc. cit.*) in discussing the experiments of Schultze (*cf. infra*) makes the following statement concerning certain of that author's results: "Das Verhältnis der beiden Geschlechter war vielmehr bei strengster Inzucht (Paarung nur mit Bruder, Enkel, Urenkel, Vater und Grossvater) ein sehr verschiedenes, ja in einigen Fällen

[1] The material in this chapter, by Maud DeWitt Pearl and Raymond Pearl, originally appeared with the title "On the relation of race crossing to the sex ratio," in *Biol. Bull.*, vol. 15, pp. 194–205, 1908. The values of the probable errors given in the original were inadvertently in error. They have been corrected and the text correspondingly altered in the present reprint.

[2] *Carnegie Institution of Washington, Publication No. 52*, 1906.

[3] Müller, R., *Biologie und Tierzucht.* Gedanken und Tatsachen zur biologischen Weiterentwicklung der landwirtschaftlichen Tierzucht. Stuttgart (Ferd. Enke), 1905, pp. 96.

kamen sogar in der dritten Geschlechtsfolge, *ganz in Gegensatze zu der älteren Annahme*,[4] überwiegend weibliche Nachkommen zur Welt."

Investigations systematically directed towards determining in what way and to what extent either hybridizing or inbreeding affect the sex ratio are very few in number. Davenport (*loc. cit.*), from a tabulation of the sex of 377 fowls reaches the conclusion already stated regarding the influence of hybridization. Schultze[5] has studied in mice the effect of inbreeding of various degrees including the closest "Inzestzucht" on sex determination, and reaches the conclusion that in general it has no effect.

The search for factors which may determine or influence sex is being actively prosecuted by experimental biologists. Any data tending to throw light on the significance of any supposed sex-influencing factors can but be welcome. The quotations from the literature which have been given suffice to indicate that the character of a mating must at least be accorded the place of a "supposed" sex-influencing factor. It is the purpose of the present chapter to exhibit and discuss certain data which have a direct and definite bearing on the question of the significance of this factor in the case of one organism, namely, man.

The data which form the basis of this study are extracted from the published vital statistics of the city of Buénos Ayres. For many years past this city has maintained an elaborate system of municipal statistics. Indeed its system might in many respects well serve as a model. The statistics of but few other cities or countries surpass those of Buénos Ayres in completeness and accuracy. These records are published in annual volumes. The statistics of births given in these volumes are particularly detailed. Among other matters of general biological interest there is given each year a table setting forth the number of births occurring in the year covered by the volume, classified in such way that it appears for each child born whether it was (*a*) male or female, (*b*) legitimate or illegitimate, and (*c*) what was the nationality of each of its parents. Furthermore it should be said of these statistics that they are *registration* figures and not *census* returns. That is to say, they are definite *records* of events, each event being recorded when it happens, not more or less inaccurate counts made a long time after the event. Of the substantial accuracy of these figures there is no doubt.

As is well known, Buénos Ayres is a city having a population which is racially very heterogeneous. For a decade and more past there has been

[4] My italics.—R. P.

[5] Schultze, O., Zur Frage der geschlechtsbildenden Ursachen. *Arch. mikr. Anat.*, Bd. 63, Heft 1, 1903.

a large Italian immigration. Also there has been extensive Spanish immigration. Representatives from other nations have come in in smaller numbers. From the statistics of birth above alluded to it is possible to determine what has been the sex of the offspring of each of these racial groups in pure matings and when crossed with native Argentine stocks. For the purpose of the present study the birth statistics of the ten years 1896–1905 inclusive have been used. The following matings have been considered:

Argentine	♂	Argentine	♀
Italian	♂	Italian	♀
Spanish	♂	Spanish	♀
Italian	♂	Argentine	♀
Spanish	♂	Argentine	♀

Data are available for other matings but it has not seemed advisable to deal with any yielding less than 8000 offspring in the ten years. The inquiry has been further limited to legitimate births, because of the uncertainty which must always exist in the great majority of illegitimate births as to whether the putative father is the actual one. With these restrictions the number of separate offspring dealt with in this study approaches a quarter of a million (exactly 219,516).

These statistics have been studied with the purpose of obtaining answers to the following questions:

1. Is there a tendency towards an excessive production of offspring of one sex (either male or female) in cross as compared with pure matings, among the human racial stocks under consideration?

2. If such a tendency appears to exist is it (*a*) uniformly shown in all matings considered, and (*b*) numerically great enough in amount to be considered significant when tested by probable errors?

DATA

The raw material is set forth in table 22. The figures are extracted from volumes VI to XV, inclusive, of the *Annuaire statistique de la ville de Buénos Ayres.*[6]

It is at once apparent that these statistics show essentially the same relation of the sexes as that usually found when large numbers of human births are examined, namely, a preponderance of males. The extent of this preponderance may be shown best by putting the data in the form of sex ratios. In this study the sex ratio will be taken as the number of

[6] Published by the Direction générale de la statistique municipale, Buénos Ayres.

males to each 100 females. The sex ratios deduced from the totals of table 22 and their probable errors are given in table 23. It does not appear to be necessary or advisable to deal with the single years separately.

TABLE 22

Sex distribution of legitimate births. Raw data

YEAR	NATIONALITY OF PARENTS									
	Argentine ♂ / Argentine ♀		Italian ♂ / Italian ♀		Spanish ♂ / Spanish ♀		Italian ♂ / Argentine ♀		Spanish ♂ / Argentine ♀	
	♂♂ Born	♀♀ Born	♂♂ Born	♀♀ Born	♂♂ Born	♀♀ Born	♂♂ Born	♀♀ Born	♂♂ Born	♀♀ Born
1896	1,597	1,654	5,326	5,455	1,814	1,695	939	932	349	411
1897	1,722	1,712	5,740	5,499	1,767	1,728	1,060	968	431	394
1898	1,922	1,773	5,765	5,703	1,805	1,695	1,152	984	431	420
1899	1,980	1,945	5,770	5,743	1,887	1,790	1,168	1,100	478	417
1900	2,038	1,950	5,070	5,620	1,809	1,784	1,126	1,064	458	411
1901	2,163	2,099	5,923	5,771	1,879	1,806	1,178	1,192	461	421
1902	2,189	2,100	5,736	5,597	1,837	1,781	1,204	1,189	463	438
1903	2,277	2,200	5,341	5,133	1,780	1,702	1,214	1,127	467	448
1904	2,352	2,368	5,419	5,240	1,952	1,723	1,345	1,215	521	460
1905	2,533	2,317	5,507	5,409	2,093	1,940	1,294	1,277	516	468
Totals.....	20,773	20,118	55,597	55,170	18,623	17,644	11,680	11,048	4,575	4,288

TABLE 23

Males to 100 females from totals of table 22

MATING	SEX RATIO	MATING	SEX RATIO
Argentine ♂ × Argentine ♀	103.26 ± 0.69	Italian ♂ × Argentine ♀	105.72 ± 0.95
Italian ♂ × Italian ♀	100.77 ± 0.41	Spanish ♂ × Argentine ♀	106.69 ± 1.53
Spanish ♂ × Spanish ♀ ..	105.55 ± 0.75		

Regarding the probable errors of the sex ratios we have the following, from well known principles. If among n births of both sexes Z is the number of male births to each 100 female births, then

$$\text{P.E.}_Z = 67.449\ (1 + Z/100)\ \sqrt{Z/100n} = \chi_1\ (100 + Z)\ \sqrt{Z/100},$$

where χ_1 is the value of $0.67449/\sqrt{n}$, published in Pearson's *Tables for Statisticians and Biometricians*. Applying this formula we get the results shown in table 23.

From this table the following points are to be noted:

1. The number of males to 100 females varies between approximately 101 and 107 in the different matings. There is an excess of males in every case. Further, except for Italian ♂ Italian ♀ this excess is significant in amount as is indicated by the probable errors. The present statistics agree with other large collections of data regarding the sex ratio of human births. There appears to be no doubt that a tendency towards the production of a greater number of males than of females is normal for Caucasian races at least.

2. The sex ratio is in each case higher for the cross matings than for the pure. That is, there are more males per hundred females produced when the parents are of different racial stocks than when they are of the same.

The answer to the first question propounded above then is that there is a definite tendency towards an increased production of male offspring in cross as compared with pure matings in the data here considered. Further, it appears that within the limits of the present material this tendency is uniformly exhibited in all the matings.

Attention may next be turned to the second part of the second question, which may now be put as follows:

Is the excess of male births in cross as compared with pure matings numerically great enough to be considered significant in comparison with the probable errors involved? The evidence on this point is presented in table 24, which compares the sex ratio for each cross mating with that for each of the two pure matings related to it. The last column of the table gives the ratio of the difference in each case to the probable error of the difference. In interpreting this last column it will be remembered that a difference which is three times as large as its probable error is to be regarded as very probably significant and if more than four times certainly so; a difference which is between two and three times its probable error is probably significant; while a difference less than twice its probable error when taken by itself is probably not significant. In general, the technical biometrical use of the term "significant" intends to convey the idea that the odds are so great as to amount to practical certainty that a so-called "significant" result did not arise simply as a purely chance effect of random sampling, but represents a direct causal nexus between phenomena.

From this table it appears that the excess of male births in the cross matings as compared with the pure is large in proportion to its probable error in the case of Italian ♂ Argentine ♀ against pure Italian matings. In two of the other comparisons the difference is twice its probable error. In one case the difference is certainly insignificant, taken by itself.

But the four different comparisons may perhaps be regarded as independent concurrent events, and we may examine the probability that a system of deviations of the magnitude of these and *all in the same sense*, would arise by chance alone. This probability is found to be 0.000,001,4. This is an extremely small value, and taken at its face value would be interpreted as meaning that such a set of results as that shown in the last column of table 24 could only be expected to arise by chance (random assorting) about once in a million trials. But here Greenwood's[7] principle of the "probability of the alternative" obviously must be considered. The

TABLE 24

Comparison of the sex-ratios of the offspring of pure and cross matings

MATINGS	SEX RATIO	DIFFERENCE / P. E. OF DIFFERENCE
Italian ♂ × Argentine ♀	105.72 ± 0.95	
Italian ♂ × Italian ♀	100.77 ± 0.41	
Difference	4.95 ± 1.03	4.8
Italian ♂ × Argentine ♀	105.72 ± 0.95	
Argentine ♂ × Argentine ♀	103.26 ± 0.69	
Difference	2.46 ± 1.17	2.1
Spanish ♂ × Argentine ♀	106.69 ± 1.53	
Spanish ♂ × Spanish ♀	105.55 ± 0.75	
Difference	1.14 ± 1.70	0.7
Spanish ♂ × Argentine ♀	106.69 ± 1.53	
Argentine ♂ × Argentine ♀	103.26 ± 0.69	
Difference	3.43 ± 1.68	2.0

only sensible course in a case of this sort is to take the indicated result as a suggestion rather than as a proved fact, and collect more independent evidence on the point. If all such evidence is consistent and in the same direction its cumulative weight finally becomes impressive to the point of conviction.

From the present data the suggested conclusion is that *there is some evidence of a significantly greater proportionate production of males in the offspring from matings involving different racial stocks than in the offspring from matings in which both parents belong to the same racial stock.*

[7] Greenwood, M., Probability and extrasensorial channels of knowledge, *Lancet*, September 29, 1923, pp. 675–676.

DISCUSSION

This conclusion is merely a statement of probable fact. In interpreting it it remains to consider two points. The first of these is as to whether there are limitations or fallacies in the data themselves which invalidate the conclusion to which they appear to lead. The second is as to what is the meaning of the facts implied by this conclusion supposing it to be true. One cannot be too cautious in drawing conclusions from human vital statistics of whatever kind. Vital statistics notoriously abound in pitfalls. In a critical examination of the data with a view to possible criticism and interpretation the following points suggest themselves:

1. That the material is not sufficiently extensive. It might conceivably be maintained that if a larger number of births were to be dealt with they would show a different result. For two reasons such a consideration appears to have little weight. In the first place the number of births included in the statistics is extremely large as measured by biological standards. The statistics include upwards of 200,000 births. In the second place the probable errors of the sex ratios indicate that the *combined* odds are large against such a consistent system of differences as that shown in table 24, arising fortuitously. In this connection it may be said that the work was begun in the first instance with the statistics for three years (1903, 1904 and 1905) only. The figures for these years led to exactly the results which have been shown above. The figures for the seven previous years were then taken into the calculation to see whether they would confirm or reverse these results. That they confirm them is clear.

2. That the inclusion of living births only in the statistics influences the result. That statistics of sex should theoretically include still-born as well as those born living is obvious. The still-born would have been included in the tables had it not been for the fact that the original material was tabulated in such way as to render it impossible to include them. A little consideration shows, however, that the absence of still-born does not sensibly affect the conclusion drawn from the present statistics. It is a well-known fact that among still-born children the proportion of males to females is very much greater than among living born. It does not seem necessary to cite evidence of this fact; all large collections of birth statistics show it. Pains have been taken to make sure that the records of still-born in Buénos Ayres form no exception to the general rule.

Now there are three possibilities respecting the distribution of still-born young among the offspring of the cross and pure matings discussed in this chapter. These are:

a. That still-births are distributed *pro rata* among cross and pure matings. This is the most probable supposition. It would be expected on general grounds that in the long run there would be substantially the same number of still-births among a given total number of births whether this total originated from cross or pure matings.

b. That a relatively larger number of still-births originate from pure than from cross matings.

c. That a relatively larger number of still-births originate from cross matings than from pure.

It being a fact that still-births show a high sex ratio it is evident that a distribution of such births in accordance with (*b*) could alone tend to reverse the conclusion reached from statistics which leave these births out of account. In case they were distributed as in (*a*) or (*c*) their inclusion would simply make more pronounced the results found in their absence. It appears highly probable on general grounds that if still-births are not proportionately distributed among cross and pure matings there is somewhat more likely to be an excess of such births arising from cross (*i.e.*, according to (*c*)) than from pure matings (*i.e.*, according to (*b*)). It is hardly conceivable that there could be a steady tendency for a sensibly greater number of still-births to occur when both parents are of the same nationality than when they are of different nationalities. If this be granted then it must also be granted that the non-inclusion of still-births in the present statistics cannot be adduced as an explanation of the observed preponderance of males in the offspring of cross matings.

3. That a different age distribution of the parents in cross as compared with pure matings may account for the observed preponderance of male births from such matings. In a population such as that here dealt with it is undoubtedly true that the males in the cross matings (being for the most part probably immigrants) are on the average somewhat older than those in the pure matings. It might conceivably be contended that this greater average age of the male parent was the cause of the excessive production of male offspring in the cross matings. To make such a contention, however, would simply be to affirm belief in Sadler's "law"[8] or some variant of it which holds that the relative age of the parents is causally related to the sex ratio of the offspring. In regard to this matter it need only be said that Sadler's theory has been abandoned by most recent students (both from the biological and from the demographic side) of the

[8] *Cf.* Geddes and Thomson, *The Evolution of Sex*, or any of the standard works on vital statistics for an account of this law.

problem of sex simply for the reason that nothing remotely approaching conclusive evidence has ever been brought forward in its support.

4. That the individuals in the cross mating are exposed to environmental influences different on the average from those acting on the individuals in pure matings and that the differences in the sex ratios of the offspring of these two groups are the result of these environmental differences. This possible explanation obviously needs careful consideration. So far as broad environmental factors such as climate are concerned there can be no differential effect on the sex ratio for the two groups since all the statistics are derived from the population of a single city. In a general sense all the individuals live in the same environment. But there is a possibility of a difference between the different groups in regard to the complex of environmental factors which are collectively implied in "social status." It is conceivable that on the average the Italian-Argentine families are of different social status than Italian-Italian or Argentine-Argentine families in the same city. Differences in social status imply differences in nutrition, in housing and in other physical conditions of existence. Some one or all of these things might conceivably be held to affect the sex ratio in the manner observed. In considering this point it needs to be held clearly in mind that there are two distinct questions involved. These are: (*a*) Is there any conclusive evidence that there does exist as a matter of fact any uniform average difference in the social status of individuals in cross as compared with pure matings? And (*b*) granting that such an average difference does exist what evidence is there that it would produce the observed effect on the sex ratio? To the first of these questions it is difficult to get any answer. Careful study of all the available demographic statistics of Buénos Ayres has failed to yield any conclusive evidence on the point. The probability appears to be, however, that if any difference at all exists in the social status of the two groups it is in the long run (or on the average) not marked in degree. Further it appears probable that whatever difference does exist is in the direction of a lower social status in the case of the cross matings.

Regarding the influence of such a difference (if it exists) on the sex ratio it seems probable that it would have very little or no effect. Punnett[9] made a very careful study of just this point for certain elements of the population of London. He found that in the classes of lower social status more females than males are born, and *vice versa*, but concludes in general that parental nutrition has no sensible influence

[9] Punnett, R. C., On nutrition and sex determination in man, *Proc. Camb. Phil. Soc.*, vol. 12, 1903.

on the sex ratio. Morgan[10] reviewed the literature on the subject and reached the following conclusion (p. 385): "If nutrition were really a factor of any importance in sex determination, it is surprising to find so little difference under apparently very favorable and unfavorable conditions. It seems much more probable that if the nutrition affects in any way the proportion of the sexes, it does so indirectly by elimination, and not by determining either the sex of the embryo or of the egg." Further on Morgan says in discussing Geddes and Thomson's theory of sex (p. 388): "If, on the other hand, the determination of sex is supposed to be due to the nourishment of the embryo, the best ascertained facts, both experimental and statistical, are opposed to the hypothesis." Taking all these points into consideration it seems very doubtful, to say the least, if the observed excess of males in the cross matings has its explanation either in whole or in part in differences in the environmental complex implied by "social status." However, in the absence of more complete and definite statistical data regarding the point one cannot be dogmatic in asserting such a conclusion.

If none of the suggested factors can reasonably be held to afford an explanation of the facts regarding the sex ratio shown by the present statistics how are these facts to be interpreted? All that can safely be asserted is that the present statistics, within their limits, suggest that there may be a definite relation between the character of the mating and the magnitude of the sex ratio. Is this a *post hoc* or a *propter hoc* relation? The data themselves do not *conclusively* demonstrate which it is. Nor does it seem probable that statistics of human births alone can ever settle this question. It is one which demands experimental analysis. The chief difficulty involved in maintaining that there is a causal relation between the character of the mating and the sex ratio lies in the lack of knowledge as to what could be the physiological mechanism by which the causation was effected. In a way the phenomenon appears somewhat analogous to the well-known phenomenon of xenia observed in plant breeding, differing in that here the character influenced is sex rather than some purely morphological feature of the organism.

In conclusion it should be said that the data presented in this chapter are not put forth as in any way final or conclusive. They require confirmation from other sources and *experimental* analysis. Within their limits, they lead to a definite and significant conclusion as to fact. In so far they contribute to the discussion of the general problem of determination of sex.

[10] Morgan, T. H., *Experimental Zoölogy*, New York, 1907, pp. xii + 454.

SUMMARY

Statistics of over 200,000 human births extending over a period of 10 years in the city of Buénos Ayres show that the proportion of males to females is in every case greater when the parents are of different racial stocks than when they are of the same. In the data are involved three racial stocks in pure and cross matings. The preponderance of males in the offspring of cross matings appears not to be capable of explanation as the result of environmental or demographic influences. Experimental investigations are necessary in order to reach adequate explanations of such statistical facts regarding sex ratios as are set forth in this chapter.

In 1919 Little[11] reported statistics from the records of the Sloane Lying-in Hospital which entirely confirm the conclusions reached in this chapter. They have further the advantage of being more reliable individually than any general vital statistics can possibly be.

[11] Little, C. C., Some factors influencing the human sex-ratio, *Proc. Soc. Exper. Biol. Med.*, vol. 16, pp. 127–130, 1919.

CHAPTER IV

The Sex Ratio among the Jews[1]

It has been suggested[2] that in cattle the proportionate number of males born tends to increase as the time of coitus becomes later and later in the oestrous period. Thus, putting all the available trustworthy data together, we have the following results:[3]

TABLE 25

Showing the sex ratio in relation to time of service

TIME OF SERVICE	SEX OF YOUNG		♂♂:100 ♀♀
	♂	♀	
Early in heat	134	178	75.3
Middle of heat	67	58	115.5
Late in heat	77	44	175.0
Totals	278	280	

It will be perceived that these figures, so far as they go, appear to give support to the theory of Thury[4] regarding sex determination. According to this theory ova which are at the time of fertilization over-ripe, or "stale," will give rise to a preponderant number of *male* young. If we may suppose, as there is some warrant for doing, that the ova fertilized late in heat are, in cattle, of a somewhat greater age (measured from the time of ovulation) than those fertilized early in heat, the results obtained accord fully with the theory. But leaving this point entirely out of account, it is indicated by the figures presented, that in cattle there is a definite relation between the proportion of the sexes born and the time at which the ova are fertilized with reference to the period of oestrus.

[1] The paper which forms the basis of this chapter, by R. P. and Redcliffe N. Salaman, originally appeared with the title "The relative time of fertilization of the ovum and the sex ratio amongst Jews," in *Amer. Anthropol.* (N.S.), vol. 15, pp. 668–674, 1913. It is here reprinted without alteration.

[2] Pearl, R., and Parshley, H. M., Data on sex determination in cattle, *Biol. Bul.*, vol. xxiv, pp. 205–225, 1913.

[3] *Loc. cit.*, p. 218.

[4] Thury, M., *Ueber das Gesetz der Erzeugung der Geschlechter bei den Pflanzen, den Thieren und dem Menschen*, Leipzig, 1864.

In his original publication Thury cited as evidence in support of his views the fact that statistics of sex show a relatively larger proportion of males to females among the Jews than in the general population of other races where they are living.

Thury's work has given rise to a number of references to the same phenomenon. Darwin,[5] quoting Thury,[6] expresses himself as much surprised at the figures given, and refers to those given for Prussia 113, Breslau 114, and Livonia 120, as compared with 104 for the non-Jewish populations.

Lagneau[7] considered the preponderance of males might be ascribed to the laws of separation practised by observant Jews, whilst Nagel[8] ascribes it to the greater care Jewish mothers take of their health, and the smaller number of illegitimate births amongst them, whilst others have ascribed it to the fact that Jews are essentially town dwellers and marry early. J. Jacobs[9] is inclined to regard the more striking differences as due to faulty statistics.

Fishberg[10] has recently discussed the problem afresh. Quoting Nichols[11] he states that whilst the sex ratio for seven million births in Europe generally is 1057:1000, the more southerly parts and the less cultured, such as Bulgaria, Greece, and Roumania, show a decidedly higher proportion of male births. In the Mussulman population of Algiers the proportion rises to 1191:1000.

These facts at once suggest that the proportion of males is higher in those countries where the birth of a male child is valued over that of a female, and further suggest that the extraordinary ratios found are probably due to the negligence of parents in recording the births of their daughters. An analysis of the Jewish birth statistics in various Russian centers does much to confirm this view. In eastern Europe the midwives and rabbis are supposed to report the births to the authorities; now amongst Eastern Jews the birth of a son is welcomed with more enthusiasm than that of a daughter. Moreover the ceremony of circumcision and the consequent festivities are events which cannot be overlooked by the rabbis.

[5] Darwin, *Descent of Man*, 2d ed., p. 243.

[6] Thury, *La loi de production des sexes*, p. 25, 1863.

[7] Lagneau, *Du Denombrement de la Population de Paris*, 1882.

[8] Nagel, Der hohe Knabenüberschuss der Neugeborenen der Jüdinnen, *Statistische Monatschrift*, p. 138, 1884.

[9] J. Jacobs, article "Births," *Jewish Encyclopædia*, vol. iii, p. 225.

[10] Fishberg, The Jews: A study of race and environment, *Contemp. Sci.*, 1911.

[11] Nichols, Numerical proportion of sexes at birth, *Memoirs Amer. Anthropol. Assn.*, vol. i, p. 247, Lancaster, Pa., 1907.

In Russia there is a further reason for the greatest accuracy in the registration of the Jewish male births. As is well known, Jews in Russia are subjected to a system of persecution which reaches from birth to death. The Jew has at all times the greatest difficulty in acquiring a passport, but if in addition his registration is out of order and he is unable to prove his identity his position is rendered so much the more precarious, while at the same time he lays himself open to all sorts of irregular demands on the part of the military officials. Fishberg is confident, and it would appear with good reason, that faulty registration is at the bottom of the anomalous Jewish birth ratio. Thus in 1893 the Jewish birth ratio was 1459:1000, but in 1901, when persecution was once more firmly established, the ratio was 1295:1000, the lower value being due to the better registration of female births.

The comparison of birth ratios of neighboring places lends much strength to the same argument. Thus in 1897 the Russian-Jewish birth ratio was 1331:1000. But in detail it was most divergent. For Taurida the ratio was 1016, but in Wilna 1774:1000. Again, Courland and Wilna are close to one another, yet their ratios are 1154 and 1774 to 1000 respectively. Again, in Prussia, 1893–1902, where statistics are naturally more accurate, the proportion of males amongst Christians was 1059:1000, and amongst Jews 1062. In Austria in 1900 it was 1068 amongst Christians and 1078 amongst Jews. In both these cases the difference is minimal.

Fishberg remarks that if the number of male births (in Russia) was genuinely so greatly in excess of female, then one would expect a corresponding excess in the returns for children of one year of age, but here we find for 1897 the ratio 1042:1000 given. The normal excess in the mortality of boys would not explain the great difference between this ratio and that given for the Jewish birth ratio, and one is forced to the conclusion that the data are untrustworthy.

Notwithstanding the probability that the statistics on which Thury and others have based their conclusions are at fault, the suggested explanation of Thury is of so much interest that it has seemed to us worthy of further investigation.

It will be remembered that Thury and Lagneau suggest that the separation (*niddah*) regulations of the Jewish women are the determining cause of the unequal sex ratio. These regulations are as follows:[12]

No connubial relations are allowed—

(a) For at least 24 hours before the expected catamenia.

[12] *The Religious Duties of a Jewish Wife*, London, published by Jacob Dickson.

(b) During the period—however transient the flow may be, it must for ritual purposes be considered as enduring at least 5 days.

(c) For a further period of 7 days from the 5th day after the commencement of the catamenia, or if it should continue longer than that period from the day on which every sign of discharge has disappeared.

These regulations are no doubt in a general way fairly faithfully followed amongst Eastern Jewesses, yet it is obvious that in mass statistics collected from the general Jewish population on the sex ratio one would not be warranted in assuming that the code given was universally or even generally applied.

To determine the influence on the sex ratio of the code, something more in the nature of an experiment was necessary. This, owing to the kindness of Dayan[13] A. Feldman, B.A., we have been able to obtain. Dayan Feldman is intimately acquainted with the life of observant Jews in the east end of London and occupies a unique and honored position in London Jewry. He has compiled for us a list of the children of 57 families in which he can with great confidence state that the laws of separation are strictly and consistently carried out in each case. Moreover in the great majority of the families here given the mothers are now beyond the child-bearing age. The parents of all the families enumerated here are Russians by birth, which renders the results obtained the more striking when one compares them with the Russian data already discussed.

In the following table we have, therefore, statistics which on the one hand are accurate, and on the other are capable of throwing definite light on the influence, if any, of the separation customs of the Jews on the sex ratio of birth:

From this table the following points are clear:

1. The families are, with few exceptions, large. The mean number of children per family, including all families in the table, is 7.28. Leaving out of account the seven small families at the end of the table, the mean number of children per family is 8.04. The fact of such relatively high fecundity in these matings adds greatly to the value of the data.

2. There is no unusual or marked preponderance of male births in these families. Taking all 57 families the sex ratio is 1054♂♂ : 1000♀♀. Leaving out the seven small families at the end of the table, too small to be of any value in the present connection, the sex ratio is 1041♂♂ : 1000♀♀. These ratios do not significantly differ from each other, nor from the ratio for the general population of England of the same period.

[13] A *dayan* is a Jewish ecclesiastical official who occupies a position which may be described as analogous to that of a judge and a bishop combined in one.

In 1910 the Registrar's return gave a sex-ratio of 1040♂♂ : 1000♀♀.[14] In the Census for 1911 the sex-ratio was 1041♂♂ : 1000♀♀,[15] whilst the children under one year are as 1022 to 1000.

TABLE 26

Dayan A. Feldman's list of families in which the Jewish separation customs are strictly adhered to

FAMILY	♂♂	♀♀	TOTAL CHILDREN	FAMILY	♂♂	♀♀	TOTAL CHILDREN
1	5	5	10	29	3	2	5
2	6	2	8	30	8	5	13
3	4	9	13	31	6	4	10
4	7	4	11	32	3	3	6
5	3	1	4	33	4	7	11
6	7	7	14	34	1	7	8
7	2	3	5	35	4	2	6
8	7	6	13	36	5	1	6
9	3	5	8	37	3	0	3
10	4	4	8	38	2	2	4
11	2	2	4	39	3	3	6
12	1	7	8	40	4	4	8
13	5	2	7	41	4	6	10
14	6	3	9	42	3	1	4
15	9	2	11	43	5	7	12
16	4	4	8	44	3	3	6
17	5	5	10	45	3	2	5
18	5	4	9	46	5	3	8
19	5	5	10	47	4	4	8
20	5	4	9	48	4	4	8
21	2	7	9	49	5	4	9
22	2	6	8	50	0	5	5
23	4	2	6	51	0	1	1
24	3	5	8	52	1	1	2
25	6	4	10	53	1	1	2
26	3	6	9	54	2	0	2
27	5	2	7	55	1	0	1
28	3	2	5	56	1	1	2
				57	2	1	3
Totals					213	202	415

While the numbers involved here are statistically small, they have some significance, we think, because of their accuracy respecting the point to

[14] *Seventy-third Annual Report of the Registrar General of Births, Deaths and Marriages in England and Wales* (1910), published 1912, p. xxiii.

[15] *Census of England and Wales*, 1911, vol. vii, pp. vi and xlix.

be tested. If the time of fertilization of the egg relative to the menstrual period had any influence in the determination of sex or in the modification of the sex ratio (such as is observed in *general* Jewish statistics), this influence would certainly be expected to make itself apparent in the present data. The families are large, and the records partake more of the character of definite experimental records than of ordinary sex-ratio statistics. More accurate and precise data than these here given it will probably be impossible to obtain for man regarding this particular point under discussion.

One would seem justified in concluding that:

a. There is no evidence that in the human race the time of fertilization of the egg relative to the catamenial period has any influence on the sex-ratio exhibited by the offspring.

b. The higher male sex-ratio shown by the general Jewish statistics, if not entirely due to faulty registration, must owe its origin to other factors than the time of fertilization of the egg.

The present results still leave entirely open the question of the metabolic condition (relative staleness, etc.) of the germ cells at the time of fertilization as a possible factor in the influencing of the sex-ratio in man.[16] The distribution of ovulation over the inter-menstrual period in the human female is so wide as to preclude any possibility of forming any judgment as to the relative age of discharged ova, on the basis of the time of menstruation.

[16] For discussion of this matter in other forms, see Pearl and Parshley, *loc. cit.*

PART II

BIOLOGICAL ASPECTS OF VITAL STATISTICS

CHAPTER V

Congenital Malformations[1]

It has been well said[2] that "the whole problem of evolution is a problem in vital statistics—a problem of longevity, of fertility, of health and of disease." While the truth of this standpoint is at the present time rapidly becoming recognized by biologists, it is only within a comparatively short time that such has been the case. A great deal of work has yet to be done in the way of analysis, from the view-point of the evolutionist statistician, of vital statistics already collected and available in the form of census and registration returns. An immense mass of biological material is presented in such returns. It is with a small portion of this material that I have dealt in this study.

The purpose of this chapter is to present an attempt at an analysis, by modern statistical methods, of certain biological aspects of the death-rate in man due to congenital malformations. The category "congenital malformation" is included among the causes of death in all the systems for the classification of vital statistics. It forms Title No. 159 of the detailed "International Classification of the Causes of Death," and includes according to this classification[3] the following specific causes of death: Chronic hydrocephalus, congenital hydrocephalus, congenital heart disease, malformation of heart, morbus ceruleus, patent foramen ovale, persistence of foramen of Botal, persistence of foramen ovale, anaspadias, anomaly, arrest of development, atresia of rectum (congenital), cleft palate, congenital abnormality, congenital clubfoot, congenital deformity, congenital eventration, congenital spina bifida, cryptorchid, ectopia, encephalocele, epispadias, exomphalos, harelip, hydrorachia, hydrorachitis, hypospadias,

[1] This paper was originally published, under the title "On the mortality due to congenital malformations, with especial reference to the problem of the relative variability of the sexes," in an obscure medical journal called *Medicine* (November, 1903) which I believe long ago passed out of existence. It was published in Detroit, Michigan. This was the writer's first paper on vital statistics, in the technical sense. It is here reprinted in its original form, except for some corrections of minor errors in the arithmetic which in no way affected the results. They bear eloquent testimony to the fact that in those days zoölogical laboratories did not include calculating machines in their equipment.

[2] *Biometrika*, vol. i, p. 320.

[3] *Cf. Manual of International Classification of Causes of Death.* Washington, U. S. Census Office, 1902, pp. 96 and 97.

imperforate anus, imperforation of intestine, malformation, meningocele, monstrosity, occlusion of anus (infant), omphalocele, podencephalus, polydactylia, spina bifida, syndactylia, talipes equinus, talipes valgus, talipes varus, vascular nevus. The classification of causes of death used in the United States census tables, before the adoption of the International Classification, included under the title "Malformation" essentially the titles enumerated above. Since the mortality returns for all the various malformations are grouped together under one heading in the census tables, the frequency distribution obtained cannot, of course, be considered to be made up of entirely homogeneous material. However, as will be brought out later, this lack of homogeneity does not invalidate the data for our purpose.

The reason for undertaking a study of the mortality returns for this category of "congenital malformations" was that the data seemed to present some important evidence on a subject of perennial interest to biologists, namely, the question of the relative variability of the sexes. Since in the census returns the male and female mortalities are separated it is possible to compare the variabilities with reference to the morbid conditions included in our category.

It has long been a current biological dictum that the male, throughout the organic world, tends to be more variable than the female. The male element in sexual reproduction was supposed, according to this view, to make for variability, and hence for progress, while the female was held to be the conservative element, making for organic stability. This view is frequently expressed in the literature, and is apparently well-nigh universally current in biological teaching. It rests, however, on comparatively little and very doubtful evidence. The bulk of the evidence which has been adduced has been from a study of certain sorts of variation in the human species. Practically all this evidence has been collected and reviewed by Havelock Ellis in his well known book entitled *Man and Woman*. Now the character of the evidence brought forward by Ellis is, in the main, this: Statistics of the gross frequency of occurrence of various abnormal or pathological conditions in man and woman are collected, and it is shown that in a number of cases these abnormal conditions occur with greater frequency among men than among women. The conclusion is thus reached that man is more variable than woman. Now, that such evidence as this gives no adequate basis whatever for any conclusion regarding the relative variability of the sexes has been very clearly brought out by Pearson[4] in a memoir devoted to this subject. He says (*loc. cit.*, pp. 266 and 267):

[4] Pearson, K., Variation in man and woman, *The Chances of Death*, vol. i, London, 1897, pp. 256–377.

Lastly, we may note that mere frequency in a pathological condition is no true test of variability at all. One sex might be much more subject than the other to this condition, and yet be really less variable with regard to it; the less afflicted sex may suffer a much wider range of degrees of intensity, and thus actually have a greater variability. I do not assert that this is so, I merely point out that, in order to prove that men are more variable even in pathological condition than women, we require, in the first place, not statistics of the frequency of the condition in the two sexes, but of its diverse degrees of intensity in the cases where it does occur.

It was with the hope of gaining some of the sort of evidence which Pearson demands regarding variation in pathological conditions that the present piece of work was undertaken. The specific problem investigated may be stated in the form of a question as follows: Is there any evidence that, with respect to all abnormalities sufficiently great to cause the death of the individual possessing them, one sex is more variable than the other? Now since this is our problem, and since the individual malformations included in the Title No. 159 are likely to occur with something like equal frequency among males and females (with the exception of the few abnormalities affecting secondary sexual characters, such as epispadias, for example, which are practically negligible as causes of death), it will be seen that the fact that the material is not strictly homogeneous does not seriously affect it for our purposes.

MATERIAL

The material used consisted of the returns for the mortality due to malformations, from the total area of the United States, in the census year 1899–1900, as given in volume IV, part 2, of the *Reports of the Twelfth Census of the United States*. The absolute frequencies of deaths, classified according to age and sex, are shown in table 27.

It is seen at once from mere inspection of the raw material that fatal abnormalities occur with considerably greater frequency among males than among females. Yet, of course, there are considerably more males than females alive at each age period, and hence in order to get figures reliable for comparison the absolute frequencies must be reduced to rates. Consequently I have reduced these absolute frequencies to frequencies *per million of the number of males and females alive at each age period*, the population data being again extracted from the same volume of the Twelfth Census Reports. The "per million" frequencies are exhibited in table 28.

Even after the reduction of the absolute to relative frequencies per million of the population in each age group, the male mortality considerably exceeds the female; or in other words, the malformations included in these data occur more frequently in men than in women.

One of the things which strike one at first sight as most remarkable about these frequency distributions is the great range. One is accustomed to think that if congenital malformations are sufficiently great to be fatal, they will cause death in a very short time after birth, certainly in less than a year. Yet the curves are seen to extend into the old age period. Of course a part of the deaths in the higher age classes reported as due to congenital malformations are incorrect returns, yet we have no reason to believe that all are to be explained in this way.

TABLE 27

Deaths from congenital malformations (still-births excluded) from twelfth census. (Absolute frequencies)

MALE		FEMALE	
Age class	Frequency	Age class	Frequency
Under 1	1414	Under 1	1170
1– 2	19	1– 2	25
2– 3	9	2– 3	7
3– 4	4	3– 4	6
4– 5	2	4– 5	3
5–10	8	5–10	5
10–15	5	10–15	5
15–20	3	15–20	1
20–25	2	20–25	1
25–30		25–30	1
30–35	1	30–35	
35–40		35–40	
40–45		40–45	
45–50		45–50	
50–55		50–55	1
55–60	2	55–60	
60–65		60–65	
65–70		65–70	1
Total.....	1469	Total.....	1226

ANALYSIS OF THE DATA

From the biological standpoint, we evidently have in these data not only a record of the frequency of occurrence of certain classes of variations or abnormalities in the human species, but also a record of the relative intensity of variations of this sort in the two sexes. Our problem is to determine whether these data give any evidence that one sex is more variable with respect to these abnormalities than is the other.

If any conclusions of value regarding the relative variability of the sexes with respect to these fatal congenital malformations are to be drawn, it is evident that it is the variation in intensity or degree of the morbid conditions which must be studied. The age distribution of the deaths furnishes data, I think, from which determinations of the variation in degree of the malformations may be made. Considering only fatal malformations, as is the case here, it seems evident that there must be a close and definite relationship between the variation in the intensity or degree

TABLE 28

Deaths from congenital malformations (still-births excluded) from twelfth census. (Per million frequencies)

MALE		FEMALE	
Age class	Frequency	Age class	Frequency
Under 1	1455.7	Under 1	1232.1
1– 2	21.2	1– 2	28.5
2– 3	9.7	2– 3	7.7
3– 4	4.3	3– 4	6.6
4– 5	2.2	4– 5	3.3
5–10	1.8	5–10	1.1
10–15	1.2	10–15	1.2
15–20	0.8	15–20	0.3
20–25	0.5	20–25	0.3
25–30		25–30	0.3
30–35	0.3	30–35	
35–40		35–40	
40–45		40–45	
45–50		45–50	
50–55		50–55	0.7
55–60	1.7	55–60	
60–65		60–65	
65–70		65–70	1.6
Total.....	1499.4	Total.....	1283.7

of the malformations and the variation in the age at death of the afflicted persons. Specifically, the following relations must hold: (1) The greater the intensity or degree of malformation in a given person, the sooner after birth will that person die, provided of course that the malformation is of sufficient consequence ever to cause death; and (2) the greater the variation in the intensity or degree of malformation in a given group of persons (*i.e.*, the less close the concentration of the variates about the mean condition) the greater will be the variation in the ages at death of

the people in the group. If these relations obtain, and it seems evident that they must, then the data make it possible to compare the variation in the intensity of the malformations in the male and female groups, since the age distributions are given and we can compare the variation in ages at death.

In order to make this comparison I have calculated the more important constants for the male and female "per million" frequency distributions.[5]

In calculating these constants care had to be exercised because the material is given partly in five-year and partly in one-year groups. Taking a unit of five years, and separating the material into two parts, in the calculation of the "rough" moments, and not using Sheppard's correction in getting μ_2, the values for the constants shown in table 29 were obtained.

For several reasons too much reliance is not to be placed in the *absolute* values of these constants, although they serve perfectly well for the purpose of comparing male and female variability. In the first place these distributions are excessively "skew," and some correction of the moments

TABLE 29

Constants for frequency distributions of table 28

	MALE	FEMALE
Mean age at death	0.647 ± 0.037 years	0.704 ± 0.053 years
Standard deviation in age at death	2.109 ± 0.026 years	2.773 ± 0.038 years
Coefficient of variation	325.9%	393.7%

ought to be made on this account. Furthermore the values found for the means (roughly 0.6 years in both cases) are probably too high, because the assumption made in the calculations that the individuals included in the age class "Under 1" centered at 0.5 years is undoubtedly untrue. There is every reason to believe, taking into consideration the causes of

[5] For a general account of the methods used in determining the constants from frequency distributions and the meaning of these constants, see Pearl, R., *Introduction to Medical Biometry and Statistics*, 1923, Philadelphia, (W. B. Saunders Co.). For the benefit of readers of this chapter who may not be familiar with the literature of biometry, it may be stated that commonly one or both of two constants, the standard deviation and the coefficient of variation, are used as measures of the amount of variation. The standard deviation is a measure, expressed in concrete units, of the concentration of the variates about the mean, and hence an excellent measure of the effective variation. A large standard deviation indicates a low degree of concentration of the variates about the mean, and consequently high variation in the character under consideration. The coefficient of variation is simply the percentage of the standard deviation in the mean, and is used in comparing the variability of characters measured in different units.

death with which we are dealing, that these individuals should be centered at somewhat less than 0.5 years. Finally it should be remembered in considering these absolute values that United States census returns of deaths are not to be relied on for extreme accuracy, to say the least. It is evident, however, that all the sources of error mentioned will in the long run affect the constants for males and females to an equal degree, and hence will leave them just as useful for purposes of comparison.

The coefficients of variation are excessively large on account of the fact that the means fall at such a low age. In the present instance the coefficients of variation alone would give an entirely false impression as to the amount of the variation. They are included merely for the sake of completeness.

In order to bring out clearly the magnitude and direction of the differences between the constants of the male and female series, table 30 has been prepared.

TABLE 30

Differences

♂ Mean + 0.057 years	=	♀ Mean
♂ Standard deviation + 0.664 years	=	♀ Standard deviation
♂ Coefficient of variation + 67.75%	=	♀ Coefficient of variation

TABLE 31

The probable error of the difference between

♂ and ♀ Mean	=	±0.065
♂ and ♀ Standard deviation	=	±0.046

The value of all these constants is greater in the female than in the male series. Of course, for reasons stated above, the very large difference between the coefficients of variation is misleading. Considering only the means and standard deviations, it may be said that women show a slightly higher mean age at death from malformations and a considerably greater variability than men. Before any final conclusions can be drawn, however, it is necessary to determine the probable errors of these differences, in order to see whether they are significant, or merely due to chance errors. The probable errors of the differences are given in table 31.

From these probable errors it is seen at once that the difference between the means is not significant, as the probable error of the difference is greater than the difference. The difference between the standard deviations is, however, significant, because here the difference is roughly over ten times as great as its probable error.

The following conclusions may now be drawn:

1. The mean age at death from fatal congenital malformations of the sorts included under Title No. 159, of the International Classification of Causes of Death, is essentially the same in men and women. Hence it may safely be concluded that the mean intensity or degree of the malformations is not essentially greater in one sex than in the other.

2. The variation in the age at death from such malformations, as measured by the standard deviation is significantly greater in women than in men, and hence we may conclude that in intensity or degree of the malformations woman is more variable than man.

Both these conclusions, it will be understood, are based on the returns of the Twelfth United States Census Reports. Whether the conclusions are generally valid or not can only be tested by the analysis of returns for the mortality from the same causes among other peoples.

CONCLUDING REMARKS

In this case just discussed it would seem that we have an example exactly corresponding to the possible case suggested by Pearson in the extract quoted above. The sex which is more subject to the abnormalities is less variable with regard to them. The less afflicted sex is more variable.

The result obtained in this case that woman is more variable than man is in agreement with the results in other cases where the question has been investigated by reliable statistical methods. Pearson (*loc. cit.*) found that, with reference to variation in several dimensions of the human skull, while the variability was not greatly different in the two sexes, there was a slight preponderance in favor of the female. On the other hand, to take an organism lower than man, Schuster has shown that in the crab *Eupagurus Prideauxi* the male is significantly more variable than the female with respect to certain dimensions of the chela and the carapace. In explanation of this he says: "We might hazard the suggestion that the female crabs . . . . are subjected to more stringent selection than the males."

It seems to the writer that in view of the results which we already have in hand it is quite absurd to attempt to formulate any general rule that either sex is in general more variable than the other. It is a question which must be settled by careful biometrical work for each particular species living in a certain environment. Variability cannot be regarded as a fixed and constant thing. On the contrary, it is something which is influenced in every species by a variety of factors each one of which is capable of change and modification.

Finally, I should like to call attention to the fact that the mortality from congenital malformations discussed in this chapter forms an excellent example of the action on the human race at the present time of natural selection "by the elimination of unfavorable variations." A certain number of children are born with variations which are unfavorable to the procedure of normal physiological processes. Our mortality curve shows the rate and manner in which these variations are eliminated by the death of the individuals bearing them. In the face of evidence of this kind it is inconceivable how any one can say that natural selection in one form or another is not acting on man at the present time.

CHAPTER VI

Centering Infant Mortality[1]

The usual custom in tabulating census returns of mortality at different ages is to use a five-year base unit for ages above 5, and below that age a one-year unit. This method of tabulation makes the finding of the moments of the frequency distribution somewhat less simple than would be the case if all the base elements were equal. Furthermore, the age distribution of the heavy mortality of the first year of life is not given at all. It becomes a very important matter to know, at least approximately, this first year distribution when one attempts to find the moments for the whole material, because the frequency in this element must be centered at some point before one can proceed with the calculations. The mean age at death of those dying under one year must be known. It is the purpose of this chapter to present a determination of the correct centering point of this first year mortality.

Now it is evident that the larger the number of frequency elements for the mortality of the first year for which we can get data, the more accurate will be the determination of the mean, for the reason that the rate of infantile mortality changes rapidly with lapse of time after birth. It is fairly easy to get data giving the number of deaths occurring in each month of the first year, but a still finer division is desirable. Especially is this true for the first month of life. Roughly from 25 to 45 per cent of all the deaths falling within the first year occur in the first month. Of these the larger proportion occur early in the month. Fortunately the German statistics give a rather detailed age grouping of the deaths under one year. I have used in this work the Prussian statistics[2] for the years 1877 to 1895, split up into three groups as follows: 1877–81, 1882–90, 1891–95. The data are given in the form of *death-rates per 1000 born* of the same class, in the given period. Separate returns are given for male and female, and legitimate and illegitimate mortality. Up to the fourteenth day after birth the mortality for each day is recorded. The mortality of the remainder of the first month is grouped together in a single

[1] This chapter, with the title "On the mean duration of life of individuals dying within a year after birth," was originally published in *Biometrika*, vol. 4, pp. 510–516, 1906.

[2] *Statistisches Handbuch für den preussischen Staat*, Bde. I, II, and III, Berlin.

class, and from the beginning of the second month to the end of the first year after birth the rates are given by months. Still-births are, of course, excluded. A glance at table 32 will make clear the way in which the returns are sub-divided.

It is evident that the grouping here is sufficiently fine to make possible a very accurate determination of the mean age at death. The material was dealt with in the following way: the rates were treated as frequencies, except in the determination of probable errors where, of course, the absolute number of deaths was used. A standard month of thirty days was assumed: then with a unit of thirty days the first and second moment coefficients about an arbitrary axis were determined. From these the position of the mean and the value of the second moment about it were easily found. Only the "rough" second moment was calculated, as it was deemed sufficiently accurate for present purposes, and furthermore it was difficult to determine the proper corrective terms to apply in this case. In the calculations each frequency element was for practical convenience, centered at the mid-point of its range. The error made by so doing is negligible.

The results are shown in table 33.

On account of a lack of necessary data it was not possible to determine the probable errors of all these constants. The general order of magnitude of the probable errors, however, may be seen from a consideration of a single case. For the period 1882-1890 data were available from which the probable errors could be determined. I find for the probable error of the mean legitimate male mortality 1882–1890 a value of ± 0.0676 day, and for the mean legitimate female mortality ± 0.0784 day. For the illegitimates the probable errors would be of course somewhat higher, although as nearly as I am able to judge from a rough estimate the probable errors of the means for this group would not exceed ± 0.15 day. The probable errors of the standard deviations will in all cases be lower. In calculating probable errors the actual number of deaths in the given group and period was taken as n.

A number of points of general interest regarding infantile mortality are brought out by this table. Before considering the main question for which the work was undertaken some of these may be discussed.

a. The mean duration of life is uniformly greater in the case of the legitimates than in the case of the illegitimates. The legitimate males have an average excess of 14.59 days, and the legitimate females an average excess of 13.96 days. The excess is almost exactly the same for each of the three periods, although the mean changes. This uniformity is re-

TABLE 32

Deaths (excluding still-born) at the designated age per 1000 legitimate or illegitimate children born

AGE IN DAYS AND MONTHS	1877–1881 AVERAGE		1882–1890 AVERAGE		1891–1895 AVERAGE	
	♂	♀	♂	♀	♂	♀
Legitimate						
0 to 1 day	9.32	7.16	10.27	7.89	10.83	8.12
1 to 2 days	5.41	4.12	5.60	4.30	5.52	4.15
2 to 3 days	3.52	2.80	3.60	2.77	3.45	2.67
3 to 4 days	2.33	1.87	2.37	1.85	2.24	1.73
4 to 5 days	1.93	1.51	1.87	1.60	1.73	1.38
5 to 6 days	2.32	1.76	2.13	1.64	1.86	1.40
6 to 7 days	2.87	2.22	2.54	1.89	2.13	1.61
7 to 8 days	2.71	2.08	2.37	1.88	2.03	1.59
8 to 9 days	2.31	1.84	2.05	1.62	1.76	1.41
9 to 10 days	1.86	1.51	1.75	1.38	1.56	1.24
10 to 11 days	1.90	1.54	1.81	1.46	1.70	1.33
11 to 12 days	1.89	1.47	1.82	1.43	1.67	1.39
12 to 13 days	1.96	1.61	1.91	1.54	1.83	1.46
13 to 14 days	1.93	1.60	1.86	1.54	1.77	1.49
14 to 1 month*	21.73	18.90	21.97	18.98	22.14	18.80
1 to 2 months*	22.59	19.76	22.70	19.79	23.20	19.78
2 to 3 months*	18.58	15.86	18.92	16.44	19.72	16.62
3 to 4 months*	15.96	13.59	16.45	14.18	16.90	14.40
4 to 5 months*	13.30	11.26	13.99	11.93	14.15	12.13
5 to 6 months*	11.51	9.85	12.07	10.46	12.24	10.55
6 to 7 months*	10.61	9.05	11.05	9.60	11.01	9.53
7 to 8 months*	9.30	8.33	9.91	8.87	9.72	8.77
8 to 9 months*	8.74	7.96	9.22	8.47	9.18	8.20
9 to 10 months*	8.29	7.83	8.69	8.20	8.30	7.85
10 to 11 months*	7.51	7.22	7.80	7.51	7.44	6.89
11 to 12 months*	6.94	6.92	7.17	6.98	6.69	6.49
	197.32	169.62	201.89	174.20	200.77	170.98
Illegitimate						
0 to 1 day	11.48	9.37	12.18	9.62	12.82	10.27
1 to 2 days	8.10	6.37	8.05	6.23	8.06	6.24
2 to 3 days	5.28	4.06	5.50	4.32	5.54	4.25
3 to 4 days	3.54	2.96	3.79	3.14	3.69	2.76
4 to 5 days	3.32	2.77	3.37	2.85	3.08	2.48
5 to 6 days	4.09	2.97	3.65	2.85	3.35	2.48
6 to 7 days	4.54	3.58	4.03	3.29	3.51	2.65

TABLE 32—*Continued*

AGE IN DAYS AND MONTHS	1877–1881 AVERAGE		1882–1890 AVERAGE		1891–1895 AVERAGE	
	♂	♀	♂	♀	♂	♀
	Illegitimate—Continued					
7 to 8 days	4.06	3.43	3.59	3.17	3.26	2.98
8 to 9 days	3.62	3.12	3.47	2.64	3.08	2.41
9 to 10 days	3.28	2.60	3.10	2.54	2.91	2.43
10 to 11 days	3.53	2.67	3.32	2.67	3.04	2.51
11 to 12 days	3.30	2.67	3.32	2.94	3.35	2.71
12 to 13 days	3.43	3.11	3.50	2.85	3.48	2.86
13 to 14 days	3.33	3.12	3.37	2.84	3.75	2.95
14 to 1 month*	44.83	39.59	46.19	40.05	48.11	42.00
1 to 2 months*	53.39	46.48	54.03	47.77	55.03	48.96
2 to 3 months*	40.95	37.06	40.93	37.86	43.36	38.63
3 to 4 months*	31.98	29.74	33.01	30.15	33.84	30.66
4 to 5 months*	24.21	23.04	25.77	23.81	26.49	25.12
5 to 6 months*	19.61	17.97	20.12	19.22	20.49	19.96
6 to 7 months*	16.03	15.38	17.04	16.45	17.45	16.84
7 to 8 months*	12.91	13.00	13.93	13.54	14.04	13.36
8 to 9 months*	11.39	11.11	11.77	12.02	11.51	11.98
9 to 10 months*	9.35	10.00	10.55	10.49	10.33	10.06
10 to 11 months*	8.50	8.63	8.56	8.87	8.27	8.29
11 to 12 months*	7.11	7.37	7.51	8.11	7.39	7.08
	345.16	312.17	353.65	320.29	359.23	322.92

* It is assumed that these are calendar months.

TABLE 33

Mean duration of life in days of individuals dying in the first year after birth

		1877–81		1882–90		1891–95	
		Mean	S.D.	Mean	S.D.	Mean	S.D.
Legitimate	♂	111.44	103.92	113.33	101.13	112.54	100.26
	♀	116.75	106.13	118.65	105.97	117.91	104.50
Illegitimate	♂	96.81	91.80	98.64	92.47	98.08	91.41
	♀	102.86	94.19	104.94	94.89	103.64	92.92

markable, and indicates to what an extent those differences in "nurture" (both pre- and post-natal) to which the difference in mean duration of life of legitimate and illegitimate infants must be attributed are uniform in long periods of time. Associated with the low mean duration of life in the case of the illegitimate infants there is of course a high death-rate as compared with the legitimate group.

b. The illegitimate infants are markedly less variable with respect to duration of life (as indicated by the standard deviation) than the legitimate. The average difference in the case of the males is 9.88 days, and in the case of the females it is 11.53 days. To adopt the illustration which has been used by Pearson, it may be said that the marksman, Death, shoots faster and with deadlier aim at illegitimate than at legitimate infants. This lower variability in the case of the illegitimates may conceivably be the result of a more sharply selective mortality than in the case of the legitimate infants.

c. The mean duration of life of those dying within a year after birth is greater in the two later periods considered than in the first. Apparently, between 1877 and 1896 there has been a gain of about a day. Does this represent a real evolutionary tendency, or is it merely a chance fluctuation? In order to get light on this question I dealt with the mortality of each of the following years separately; 1882, '83, '84, '85, '86, '89, 1890, '91, '94, '95 and '96. These were all the years for which I could get separate data. The groups of male and female, legitimate and illegitimate were treated separately as in the other cases. The results obtained were very interesting in many particulars, but as they fall outside the scope of this discussion I shall not consider them in detail here. The general tendency from about 1884 on is for the mean duration of life to decrease, with considerable fluctuations from year to year. Thus in 1886 and in 1890 there was an unusually high duration of life. These two years explain why the 1882–90 group in table 33 shows such high means. In general, a study of these individual year data makes it clear that there is no steady tendency towards lengthening of the mean duration of life of infants under 1, *within* the period under consideration.

Another interesting point brought out by the single year records is that in the case of both legitimate and illegitimate infants, there is a definitely marked tendency for an increase in the mean duration of life in any year to be associated with an increase in the death-rate for that year. This appears to indicate that in general there is a tendency for any increase in the infantile death-rate to be the result of an increased number of deaths of older rather than younger infants (under 1). It will be noted, however, that this positive relation between death-rate and mean duration of life which appears *within* both legitimate and illegitimate groups is exactly reversed when the two groups are themselves compared. Thus in the legitimate group as a whole we have a condition of low death-rate and high mean duration of life, while in the illegitimate group as a whole the opposite condition obtains.

The single year means are given in table 34.

d. The mean duration of life is uniformly greater in the females than in the males. The real basis for the difference is not clear. It can hardly

TABLE 34

Mean age in days of individuals dying under one in the designated years. (The death rate for the same group is given in brackets below each mean age value)

YEAR	♂		♀	
	Legitimate	Illegitimate	Legitimate	Illegitimate
1882	111.71 (202.57)	96.39 (341.90)	117.89 (173.02)	104.17 (314.21)
1883	112.91 (204.94)	100.04 (356.52)	117.72 (175.92)	103.80 (322.44)
1884	114.07 (205.96)	98.86 (360.83)	119.70 (178.13)	103.09 (322.02)
1885	113.91 (197.55)	98.15 (341.24)	119.34 (169.97)	106.26 (311.06)
1886	117.15 (217.29)	101.79 (377.47)	121.46 (188.98)	108.91 (343.06)
1889	112.97 (200.00)	98.00 (363.40)	118.28 (172.90)	104.72 (330.00)
1890	114.62 (204.92)	100.53 (360.65)	122.99 (177.27)	109.05 (324.83)
1891	110.96 (196.55)	96.37 (356.66)	116.26 (166.29)	102.54 (321.75)
1894	113 61 (190.80)	98.15 (339.52)	119.23 (163.65)	104.16 (304.46)
1895	111.89 (207.18)	98.58 (376.59)	116.89 (175.36)	104.07 (337.16)
1896	111.67 (187.62)	95.85 (328.69)	116.83 (159.29)	101.79 (296.99)

be due to differing conditions of "nurture" because there is apparently no reason to suppose that in the long run the environment of a male infant differs in any marked and constant way from that of a female infant during

the first year after birth. Along with the lowering of the mean age at death in the male there is a smaller amount of variation in this character.

We may turn now to the practical question as to where the first year mortality shall be centered. The values of the mean for the different groups range between three and four months and each may be taken as the centering point of its own group. It is desirable, however, to have a single value for the total first year mortality, when males and females, and legitimates and illegitimates are grouped together. In order to obtain such a value resort was had to the plan of a weighted average of all the individual values. Several schemes of weighting were tried, but as all gave very closely accordant results, and the one to be described seemed the most logical, it alone need be detailed here. Examination of all the available data indicated that the absolute number of deaths of males under 1 stood in about the ratio of 1.25 : 1 to the absolute number of deaths of females under 1. So then the mean age at death of males and females together for each period was determined, the two contributory means being weighted in this proportion 1.25 to 1. Legitimates and illegitimates were dealt with separately. Then the mean age for the whole period 1877–95 for both legitimates and illegitimates was determined. In getting these values the three periods 1877–81, 1882–90, 1891–95 were weighted in the proportions 1 : 2 : 1 respectively. In this way was obtained the mean duration of life of legitimates and illegitimates irrespective of sex. It remained to combine into a single average the legitimates and illegitimates. I found that during the period under discussion there died, on the average, 6.556 legitimate infants under 1, to 1 illegitimate infant. Averaging, then, the legitimate and illegitimate means previously obtained, with weights of 6.556 and 1, respectively, I found for the mean duration of life of all infants dying under age 1, during the period 1877–95 in Prussia, a value of 113.14 days. With a standard month of thirty days this equals 3.771 months, or 0.3142 year. Reckoning 365 days to the year we have from the days directly the mean equal to 0.30997 year.

I conclude that the deaths occurring in the first year of life may be centered at 0.3 year with sufficient accuracy for ordinary purposes.[3]

We may turn now to two other practical problems which arise in connection with infantile mortality. These are

1. At what age shall the deaths occurring in the first month of life be centered?

[3] The error made by centering each element of the frequency distributions of table 32 at the midpoint would tend to raise slightly the mean age at death for the year. Hence it is probable that 0.3 year comes closer to the true value than 0.31 year.

2. At what age shall the deaths recorded in census returns as falling in the age period 0–5 years be centered?

For a solution of the first problem I have taken the Prussian statistics for deaths in the first month of life, during the period 1882–90 (table 32); I find the following values for the means:

Age at death of those dying before completing the first month of life

LEGITIMATE		ILLEGITIMATE	
♂	♀	♂	♀
10.82 days	11.25 days	12.41 days	12.75 days

From these results it seems reasonable to conclude that the deaths during the first month of life may be centered, with sufficient accuracy for practical purposes, at 0.3 month.

In order to test this result I have calculated the mean age at death of English infants dying under age 1, from the following statistics given in Newsholme's *Vital Statistics*.[4]

Annual death-rate per 1000 living at each month of age. Healthy districts

AGE IN MONTHS	DEATH-RATE	AGE IN MONTHS	DEATH-RATE
0	447.51	6	70.54
1	145.49	7	65.97
2	102.05	8	61.85
3	87.16	9	58.32
4	81.09	10	55.28
5	75.54	11	52.86

Centering the mortality of the first month at 0.3 month and that of each succeeding month at the mid-point I find for the mean 113.09 days, a result in very close agreement with that obtained from the German statistics.

For determining the mean duration of life of those dying within the age class "0 to 5 years" I have taken as a basis for computation the age distribution of the deaths falling in the first five years of life from the Report of the Twelfth Census of the United States, for the Registration Area. The distribution is as follows:

[4] Second edition, p. 105.

	AGE					TOTAL
	Under 1 year	1 year	2 years	3 years	4 years	
Deaths.....	102,220	25,986	12,020	7,825	5,520	153,571

Now, evidently, the mean age at death for these five groups will fall between

$$\frac{102220 \times 0.3 + 25986 \times 1.3 + 12020 \times 2.3 + 7825 \times 3.3 + 5520 \times 4.3}{153571} = 0.9224,$$

and

$$\frac{102220 \times 0.3 + 25986 \times 1.5 + 12020 \times 2.5 + 7825 \times 3.5 + 5520 \times 4.5}{153571} = 0.9893,$$

if we assume essentially the same distribution of deaths under 1 year in the American population as is shown in the Prussian.[5] The mean of these two values is 0.956 year. The true value probably lies somewhere between 0.95 year and 0.99 year. With a sufficient degree of accuracy for most cases the deaths recorded in the class "Under 5" may be centered at 1 year.

Briefly stated the results are, then, as follows:

a. The deaths recorded in the age class "Under 1 month" may in practical work be centered at 0.3 month.

b. Those recorded in the age class "Under 1 year" may be centered at 0.3 year.

c. Those recorded as "Under 5 years" may be centered at 1 year.

[5] Since the deaths in the second, third, fourth and fifth years of life clearly will not center lower than those of the first year, nor higher than the mid-point of each year.

CHAPTER VII

Mortality and Evolution[1]

I

It is the purpose of this chapter to set forth some facts regarding human mortality which appear to lead with great clarity to certain evolutionary generalizations of interest to the biologist, which have hitherto been overlooked so far as I am aware. The present fashion in the study of evolution is towards the analytical discussion of the factors. Synthetic general discussions of broad phases of organic evolution, which occupied so prominent a place in early post-Darwinian times, are now but rarely found in biological literature. This may fairly be regarded as a blessing, but perhaps not an entirely unmitigated one. While much of the general discussion of evolution of the period of fifty years ago was utter nonsense, still a view of some of the aspects of the forest may be at least occasionally stimulating, and particularly in these present days when we are accumulating such a mass of precise data about the characteristics of the trees.

It is in some ways remarkable that so little thought and interest have been given by general biologists to the phases of biology which form the working material of that branch of applied science which is roughly but still sufficiently intelligibly labelled "vital statistics." The data of human natality, morbidity and mortality, when intelligently and broadly studied, can, I am sure, throw a great deal of light on some of the deepest and most significant problems of general biology. If the facts presented in this

[1] This chapter is based upon a paper first published, under the title "Certain evolutionary aspects of human mortality rates," in the *American Naturalist*, vol. 54, pp. 5–44, 1920.

In the present reprint I have taken the opportunity of altering the paper to conform to the changes in my own views since it was originally published, and to meet certain criticisms which have been made regarding the original distribution of some of the causes of death. Also I have incorporated, at the end, an index to the biological classification of causes of death with which the chapter deals, along the lines of one originally published in *Metron*, vol. 1, pp. 92–99, 1921.

In making this study, both originally and in the present reprint, I have had the benefit in matters of pathological anatomy and embryology of the critical acumen and wide knowledge of my colleague, Dr. W. T. Howard, to whom I am greatly indebted for this help.

chapter succeed in some small degree in demonstrating that this opinion is not an entirely idle one, the purpose of this particular piece of work will have been served.

II

By an international agreement among statisticians the causes of human mortality are, for statistical purposes, rather rigidly defined and separated into something over 180 distinct causes. It should be clearly understood that this convention is distinctly and essentially statistical in its nature. In recording the statistics of death the vital statistician is confronted with the absolute necessity of putting every death record into some category or other in respect of its causation. However complex biologically may have been the train of events leading up to a particular demise, the statistician must record the terminal "cause of death" as some particular thing. The International Classification of the Causes of Death is a code which is the result of many years' experience and thought. Great as are its defects in certain particulars, it nevertheless has certain marked advantages, the most conspicuous of which is that by its use the vital statistics of different countries are put upon a uniform basis.

The several separate causes of death are grouped in the Second Decennial Revision of the International Classification, which was the one used in this study, into the following general classes:

I. General diseases
II. Diseases of the nervous system and of the organs of special sense
III. Diseases of the circulatory system
IV Diseases of the respiratory system
V. Diseases of the digestive system
VI. Non-venereal diseases of the genito-urinary system and annexa
VII. The puerperal state
VIII. Diseases of the skin and of the cellular tissue
IX. Diseases of the bones and of the organs of locomotion
X. Malformations
XI. Early infancy
XII Old age
XIII. External causes
XIV Ill-defined diseases

It is evident enough that this is not primarily a biological classification. The first group, for example, called "General diseases," which caused in 1916, in the Registration Area of the United States approximately one fourth of all the deaths, is a curious biological and clinical melange. It includes such diverse entities as measles, malaria, tetanus, tuberculosis,

cancer, gonococcus infection, alcoholism, goiter, and many other equally unlike causes of death. For the purposes of the statistical registrar it has useful points to make this "General diseases" grouping, but it clearly corresponds to nothing natural in the biological world. Again, in such part of the scheme as does have some biological basis, the basis is different in different rubrics. Some of the rubrics have an organological base, while others, as "Malformations" have a causational rather than an organological base.

Altogether it is evident that if any synthetic biological use is to be made of mortality data a fundamentally different scheme of classification of the causes of death will have to be worked out.

III

For the purposes of this study[2] I have developed an entirely different general classification of the causes of death on a reasonably consistent biological basis. The underlying idea of this new classification is to group all causes of death under the heads of the several organ systems of the body, the functional breakdown of which is the immediate or predominant cause of the cessation of life. All except a few of the statistically recognized causes of death in the International Classification can be assigned places in such a biologically grouped list. It has a sound logical foundation in the fact that, biologically considered, death results because some organ system, or group of organ systems, fails to continue its function. Practically, the plan involves the reassignment of all of the several causes of death now grouped by vital statisticians under heading "I. General diseases." It also involves the re-distributing of causes of death now listed under the puerperal state, malformations, early infancy, and certain of those under external causes.

The headings finally decided upon for the new classification are as follows:

[2] It should be clearly understood that this phrase "For the purposes of this study" means precisely what it says. I am not advocating a new classification of the causes of death for statistical use. I should oppose vigorously any attempt to substitute a new classification (mine or any other) for the International List now in use. Uniformity in statistical classification is essential to usable, practical vital statistics. Such uniformity has now become well established through the International Classification. It would be most undesirable to make any radical changes in the Classification now. I have in this paper made a rearrangement of the causes of death, for the purposes of a specific biological problem, and no other. I am not "proposing a new classification of vital statistics" for official or any other use except the one to which I here put it.

I. Circulatory system, blood, and blood-forming organs
II. Respiratory system
III. Primary and secondary sex organs
IV. Kidneys and related excretory organs
V. Skeletal and muscular systems
VI. Alimentary tract and associated organs concerned in metabolism
VII. Nervous system and sense organs
VIII. Skin
IX. Endocrinal system
X. All other causes of death

It should be emphasized before presenting the tables of detailed statistics on this new classification that the underlying idea of this rearrangement of the causes of death is to put all those lethal entities together which bring about death because of the functional organic breakdown of the same general organ system. The cause of this functional breakdown may be anything whatever in the range of pathology. It may be due to bacterial infection; it may be due to trophic disturbances; it may be due to mechanical disturbances which prevent the continuation of normal function; or to any other cause whatsoever. In other words, the basis of the present classification is not that of pathological causation, but it is rather that of organological breakdown. We are now looking at the question of death from the standpoint of the pure biologist, who concerns himself not with what causes a cessation of function, but rather with what part of the organism ceases to function, and therefore causes death. It is to be hoped that the novelty of this method of looking at the causes of human mortality will not *per se* prejudice the reader against it, to the degree at least of preventing him from examining the detailed results and consequences of such classification, which will be presented in what follows.

There will now be presented in a series of tables the statistical data as to deaths arranged in this classification. The data given are in the form of death rates per hundred thousand living at all ages from various causes of death, arranged by organ systems primarily concerned in death from the specified disease. The statistics presented are from three widely separated localities and times, viz., (*a*) from the Registration Area of the United States; (*b*) from England and Wales; and (*c*) from the City of São Paulo, Brazil. The first two columns of each table give the death rates, arranged in descending order of magnitude in the first column, for the Registration Area of the United States for the two periods, 1906–10 and 1901–05. The third column of each table gives the death rate from the same cause of death for England and Wales in the year 1914. The fourth column gives the rates for São Paulo for the year 1917. The data

for the United States Registration Area were extracted from the volume of Mortality Statistics for 1916, issued by the Bureau of the Census. The English data were extracted from the Report of the Registrar General of

TABLE 35

Circulatory system, blood and blood-forming organs

NO.*	"CAUSE OF DEATH" AS PER INTERNATIONAL CLASSIFICATION	REGISTRATION AREA, U S.A.		ENGLAND AND WALES 1914	SÃO PAULO 1917
		1906–10	1901–05		
79	Organic diseases of the heart	133.2	124.2	137.3	130.0
64	Cerebral hemorrhage and apoplexy	71.7	69.6	65.3	32.9
81	Diseases of the arteries	17.7	9.4	23.5	59.7
78	Acute endocarditis	12.2	11.2	5.1	6.5
6	Measles	10.8	9.0	24.7	1.5
7	Scarlet fever	10.6	11.0	7.7	5.4
150†	Congenital malformation of the heart	9.0	6.7	4.2	4.6‡
80	Angina pectoris	6.8	6.6	3.2	2.2
82	Embolism and thrombosis	3.9	4.2	8.9	8.3
20	Purulent infection and septicemia	3.8	6.1	1.8	22.2
142	Gangrene	3.5	4.5	4.4	2.6
4	Malaria	2.6	4.8	0.2	2.8
65	Softening of the brain	2.5	3.7	3.9	0.7
85	Hemorrhage and other diseases of the circulatory system	1.6	2.8	0.6	2.4
53	Leukemia	1.5	1.2	2.0	2.0
77	Pericarditis	1.3	2.1	1.2	1.1
54	Anemia, chlorosis	1.0	0.5	6.4	3.7
83	Diseases of the veins	0.6	0.6	1.0	0.7
84	Diseases of the lymphatics	0.3	0.2	0.9	0.2
116	Diseases of the spleen	0.2	0.3	0.2	0.4
22	Anthrax	0.2	0.1	0	0
16	Yellow fever	0	0.3	0	0
15	Plague	0	0.1	0	0
21	Glanders	§	§	0	0
2	Typhus fever	§	§	0	0
3	Relapsing fever	§	§	0	0
11	Miliary fever	§	§	0	0
Totals		295.0	279.2	302.5	289.9

* The numbers in this column in this and the following tables are the numbers of the several causes of death in the International Classification, Second Decennial Revision.

† In part.

‡ The São Paulo statistics do not separate congenital malformations. This is the total rate.

§ Less than 0.1 per 100,000.

England and Wales for 1914. The São Paulo rates were calculated from data as to deaths and population given in the *Annuario Demographico* of São Paulo for 1917.

Nine of the items in table 35, namely, items 77 to 85 inclusive, are those of rubric III of the International Classification, "Diseases of the circulatory system." The other items of table 35 require some special explanation.

No. 64, "Cerebral hemorrhage and apoplexy," was originally placed with the nervous system in the biological classification. I was never entirely satisfied with this disposition of this important cause of death, and I am now wholly convinced that it was an error. It plainly belongs with other hemorrhages here.

Again, softening of the brain is really a necrosis of brain tissue resulting from a cutting off of its nourishment by stoppage of the circulation, which in turn may be due to arteritis, thrombosis, embolism or pressure. The same reasoning applies here as in the case of cerebral hemorrhage.

No. 7, "scarlet fever," appears in the International Classification under "General diseases." It is placed here in the organological classification because in the vast majority of cases of fatal scarlet fever it is the clinical form of the disease known as septic scarlatina which is responsible for the death. Spengarn[3] says that "septic scarlatina is responsible for most of the deaths." "The general condition is one of septicemia." It, therefore, seems best, on the present plan of biological classification, to put scarlet fever with the circulatory system, blood and blood-forming organs, since septicemia is the result of a breakdown and failure to function of the normal defensive serologic mechanism of the body.

On the same reasoning item 6, "Measles," has been transferred to this table from the respiratory system where it was originally placed. I now believe this to be a better disposition of the case.

The question may fairly be raised as to whether item 22, "Anthrax," should come in table 35 with the blood rather than with the skin in table 42, where it was originally. It is a difficult question and one not capable of any absolutely precise solution in the nature of the case. Most fatal cases of anthrax, if not all, are septicemias, or, perhaps better, bacteremias, due finally to failure of the defensive mechanism of the blood. Furthermore, pneumonic and intestinal forms of anthrax occur. On the whole, however, the weight of evidence seems to be that while in the majority of cases at least the organism gains its entrance and first victory through the skin, it is the biological strength or weakness of the blood as a defensive

[3] Spengarn, A., Scarlatina, in *Ref. Handbook Med. Sci.*, vol. VII, p. 658, 1916.

mechanism that determines primarily what will subsequently happen. Fortunately, the total rate from anthrax is so small as to be of no significance in any general result. The same reasoning applies to item 21, "Glanders."

The item 150 in the International Classification is entitled "Congenital malformations," and there includes the following three subdivisions: hydrocephalus, congenital malformations of the heart, and other congenital malformations. The second of these subdivisions, "congenital malformations of the heart," obviously belongs here, and is consequently included, while the other subdivisions do not.

Item 20, "Purulent infection and septicemia," is taken from "General diseases" and put here on the same reasoning as that just stated for scarlet fever.

Item 142, "Gangrene," is placed here because normally in civilian life, under the conditions which prevailed when these statistics were taken, most fatal gangrene is due to impairment of the circulation as a primary cause. The arteries become occluded either from endarterial inflammation, due either to frank infection, or to various somewhat obscure causes producing local obliterative endarteritis, or to trauma, or to thrombosis or embolism, especially in association with cardiac disease. Again some cases of gangrene, in the sense under consideration here, are doubtless due to extensive phlebitis and primary thrombosis of veins. In any case it is a part of the circulatory system which breaks down, and therefore we are warranted in placing this disease in table 35.

Item 4, "Malaria," is fundamentally a disease of the blood, and hence is placed here from "General diseases."

All the evidence that the pathological anatomist has leads to the view that yellow fever, typhus fever, relapsing fever and miliary fever are blood diseases. They have the lesions of septicemias, or are transmitted by biting insects, or both.

Items 53 and 54, "Leukemia" and "Anemia, chlorosis," represent breakdowns of the blood or blood-forming organs of the body. They are taken from class I of the International Classification.

In the International Classification item 116, "Diseases of the spleen," is placed under the general rubric of "Diseases of the digestive system." Just what the spleen has to do directly with digestion does not appear. It is primarily an organ concerned with the regulation of the blood.

Bubonic plague is ordinarily a disease of the lymphatic system. The great epidemics of fatal type are characterized by the pneumonic and septicemic forms. On the whole, it seems best to place this disease here.

It is evident from the data of table 35 that where death ensues from a breakdown of any part of the circulatory or blood systems it is preponderantly the heart itself or the arteries which are at fault. The other causes listed are of relatively minor importance. The relatively enormous rates for diseases of the arteries and for purulent infection and septicemia in São Paulo are noteworthy.

TABLE 36

Respiratory system

NO.	"CAUSE OF DEATH" AS PER INTERNATIONAL CLASSIFICATION	REGISTRATION AREA, U.S.A.		ENGLAND AND WALES 1914	SÃO PAULO 1917
		1906–10	1901–05		
28&29	Tuberculosis of lungs (including acute miliary tuberculosis)	146.8	170.7	104.5	119.8
92	Pneumonia (lobar and undefined)	103.0	125.5	57.5	59.9
91	Bronchopneumonia	40.4	32.9	50.9	103.9
9	Diphtheria and croup	22.4	29.6	16.0	9.6
10	Influenza	16.4	19.9	16.1	16.1
89	Acute bronchitis	15.2	21.4	108.7*	62.1
8	Whooping cough	11.5	10.9	21.8	9.1
90	Chronic bronchitis	11.1	15.4		3.9
94	Pulmonary congestion and apoplexy	5.6	8.6	4.5	9.4
93	Pleurisy	4.1	4.9	4.0	7.6
96	Asthma	2.9	3.7	4.9	2.8
98	Other respiratory diseases	2.8	4.3	1.7	5.2
87	Diseases of the larynx	1.7	2.3	3.2	0.9
97	Pulmonary emphysema	0.4	0.7	1.2	2.2
95	Gangrene of the lungs	0.4	0.5	0.3	3.3
86	Diseases of the nasal fossae	0.2	0.2	0.2	0.2
Totals		384.9	451.5	395.5	416.0

* Includes acute and chronic bronchitis.

For the present no attempt will be made to discuss the reasons for these differences, since the main object in this section is to get the data as a whole before the reader.

The causes of death listed in table 36 include all of those under the general heading IV, "Diseases of the respiratory system" of the International Classification, with a single exception, namely No. 88, "Diseases of the thyroid body," which goes elsewhere in the present classification. In addition, there are in table 36 four causes of death which are not included with the respiratory system in the International List. These four we may consider in detail.

Item 28, "Tuberculosis of lungs," obviously belongs with the respiratory system, in a strictly organological classification. The breakdown of the lungs as a functioning system is the biological meaning of death from pulmonary tuberculosis. This item is taken from rubric I, "General diseases," of the International Classification. Acute miliary tuberculosis has been included with pulmonary tuberculosis here, rather than as a separate item. No significant error is introduced by this procedure for two reasons: (*a*) the rate from miliary tuberculosis by itself is very small; and (*b*) probably a majority of cases of acute miliary tuberculosis have the lungs as the chief organ affected.

Item 9, "Diphtheria and croup," is again obviously a respiratory category, on the basis of organs affected. It does not seem to me to be to the point to argue that death in diphtheria is in many cases due to a general toxemia. To do so brings into prominence an aspect of the matter foreign to our present point of view. The infecting agent attacks a part of the respiratory system. If that system were in man as in the insects, lined with chitin in considerable part, presumably death from the clinical entity known as diphtheria would never occur, because the organism would not get the necessary foothold to produce enough toxin to be troublesome. It seems to me further that there is a fundamental biological difference between the cases of scarlet fever, measles, and septicemia on the one hand, and diphtheria on the other hand, which leads to the placing of the former with the blood and the latter with the respiratory system. It is apparent, of course, that the matter of the placing of diphtheria can be argued from both sides, but on the whole I incline to the view that it belongs here with the respiratory organs rather than with the blood.

Item 10, "Influenza," is so obviously respiratory as to require no discussion. The same may be said of item 8, "Whooping cough."

Table 36 brings out very clearly one important point in favor of the present classification. It is evident from an examination of the four columns of rates that the usages in respect of the diagnostic terminology of respiratory affections, especially the pneumonias and bronchitis, differ greatly in these three countries. Yet the *totals* for all respiratory system deaths are closely similar for all three countries and periods. In other words, the organological totals get rid to a large degree of one of the greatest sources of error in vital statistics, the varying terminology of disease in different regions.

The first and the fourth items in table 37 present a new angle of the problem of the classification of the causes of death which needs particular discussion. These items, "Premature birth" and "Injuries at birth"

represent a part of the items 151 and 152 of the International Classification. In the International Classification, item 151, which comes under the general heading "XI. Early infancy," has this general title "Congenital debility, icterus and sclerema (total)." This contains two separate subdivisions not numbered, the first being "Premature birth," and the second

TABLE 37

Primary and secondary sex organs

NO.	"CAUSE OF DEATH" AS PER INTERNATIONAL CLASSIFICATION	REGISTRATION AREA, U.S.A.		ENGLAND AND WALES 1914	SÃO PAULO 1917
		1906–10	1901–05		
151*	Premature birth	35.7	30.8	46.9	66.8
42	Cancer of the female genital organs	10.8	10.0	12.9	6.5
137	Puerperal septicemia	6.8	6.3	3.7	6.5
152*	Injuries at birth	6.6	5.0	2.8	2.1
43	Cancer of the breast	6.5	5.6	10.4	1.5
37	Syphilis	5.4	4.1	5.8	15.0†
126	Diseases of the prostate	3.4	2.6	4.2	0.7
132	Salpingitis and other diseases of ♀ genital organs	2.2	2.1	0.5	0.2
129	Uterine tumor (non-cancerous)	1.8	1.8	0.8	0
134	Accidents of pregnancy	1.7	1.7	1.1	0.2
130	Other diseases of the uterus	1.6	1.7	0.4	0.4
136	Other accidents of labor	1.3	0.9	1.1	0.7
140	Following childbirth	1.1	1.5	0.1	
131	Cysts and other tumors of ovary	1.0	1.3	0.8	0.2
135	Puerperal hemorrhage	1.0	1.0	1.3	1.7
125	Diseases of the urethra, urinary abscesses, etc	0.4	0.4	1.2	0.7
38	Gonococcus infection	0.3	0.1	0.2	0
128	Uterine hemorrhage (non-puerperal)	0.2	0.3	0	0
127	Non-venereal diseases of ♂ genital organs	0.1	0.1	0.2	0
133	Non-puerperal diseases of breast (except cancer)	0.1	0.1	0.1	0
139	Puerperal phlegmasia, etc	0.1	‡	0.9	0
Totals		88.1	77.4	95.4	103.2

* In part. *Cf.* text here.

† Including soft chancre (syphilis 1.5, and soft chancre 13.5).

‡ Less than 0.1 per 100,000.

"Congenital debility, atrophy, marasmus, etc." Item 152, coming under the same general head of the International Classification has the general title "Other causes peculiar to early infancy (total)." This term contains two unnumbered subdivisions, the first being "Injuries at birth," and the other "Other causes peculiar to early infancy."

The question at once arises, why should these two items "Premature birth" and "Injuries at birth" be included with the primary and secondary sex organs, since it is obvious enough that the infants whose deaths are recorded under these heads in the vast majority of cases, if not all, have nothing whatever the matter with either their primary or secondary sex organs. The answer is, in general terms, that on any proper biological basis deaths coming under either of these two categories are not properly chargeable organically against the infant at all, but should be charged, on such a basis, against the mother. To go into further detail, it is apparent that when a premature birth occurs it is because the reproductive system of the mother, for some reason or other, did not rise to the demands of the situation of carrying the fetus to term. Premature birth, in short, results from a failure or breakdown in some particular of the maternal reproductive system. This failure may be caused in various ways, which do not here concern us. The essential feature from our present viewpoint is that the reproductive system of the mother does break down, and by so doing causes the death of the infant, and that death is recorded statistically under this title "Premature birth." The death organically is chargeable to the mother.

A considerable number of cases of premature birth are unquestionably due to placental defect and the placenta is a structure of fetal origin, so such deaths could not be properly charged to the mother. On the other hand, however, they would still stay in table 37, because the placenta may fairly be regarded as an organ intimately concerned in reproduction.

The same reasoning which applies to premature births, *mutatis mutandis*, applies to the item "Injuries at birth." An infant death recorded under this head means that some part of the reproductive mechanism of the mother, either structural or functional, failed of normal performance in the time of stress. Usually "injury at birth" means a contracted or malformed pelvis in the mother. But in any case the death is purely external and accidental from the standpoint of the infant. It is organically chargeable to a defect of the sex organs of the mother. The female pelvis, in respect of its conformation, is a secondary sex character.

A practical difficulty arose from the fact that in the São Paulo statistics items 151 and 152 are not subdivided. In the case of the first of these, item 151, I have ventured to divide the total rate in roughly the same *proportion* between the two subdivisions as exists in the United States and England, namely, $\frac{3}{5}$ to premature birth and $\frac{2}{5}$ to congenital debility, etc. While this is admittedly a hazardous proceeding, it seems to me less so than to omit entirely so important a rate, which is the only other prac-

tical alternative. In the case of item 152 the total rate is so small (3.3) that no particular difference will be made whatever the basis of distribution used. Consequently, I have again divided it roughly on the basis of the American figures, calling $\frac{5}{8}$ of the total due to injuries at birth.

Table 37 also includes data which in the International Classification are distributed under three different general heads. First, "General diseases"; second, "Non-venereal diseases of the genito-urinary system and annexa"; and third, "Puerperal state." In the International List all cancers are included under "General diseases." We have taken out for inclusion here the several cancers of the primary and secondary sex organs, including item 42, "Cancer of the female genital organs," and item 43, "Cancer of the breast." Items 37 and 38, "Syphilis" and "Gonococcus infection," are also taken out of the class of "General diseases" of the International List. The immediate reason for including these diseases here is obvious, but particularly in relation to syphilis the point at once needs further discussion. As a cause of actual death, syphilis frequently acts through the central nervous system or through the heart, and the question may fairly be raised why, in view of this fact, syphilis is not included in those places. The point well illustrates one of the fundamental difficulties in any organological classification of disease. In the case of syphilis, however, the difficulty in practise is not nearly so great as it is in theory. As a matter of fact, most of the deaths from the effect of syphilitic infection on the nervous system are recorded in vital statistics by reporting physicians and vital statisticians as diseases of the nervous system. For example, it is perfectly certain that most of the deaths recorded as due to "locomotor ataxia" are fundamentally syphilitic in origin. The rate included in table 37 of 5.4 for the Registration Area of the United States in 1906–1910 for deaths due to syphilis is far lower, as any clinician knows, than the number of deaths really attributable to syphilitic infection. These other deaths, due to syphilis, and not reported under that title, are reported under the organ which primarily breaks down and causes death, as, for example, the brain, and will in the present system of classification be included under the nervous system. After careful consideration it has seemed as fair and just as anything which could be done to put the residue of deaths specifically reported as due to syphilis under table 37, primary and secondary sex organs. The rate in any event is so small that whatever shift was made could not sensibly affect the general results to which we shall presently come.

The question may be asked as to why puerperal septicemia (item 137) is included here and not with the diseases of the circulatory system and

blood on the same reasoning that general septicemia was put there. The cases seem to be essentially different. Puerperal septicemia arises fundamentally because of a failure of the reproductive system of the female to meet in a normal way the demands made upon it by the process of reproduction itself. In line with the general reasoning on which we are working in this classification, it would therefore seem that this cause of death belongs where it has been put here, with the primary and secondary sex organs. The same sort of reasoning applies to the other puerperal causes of death here included.

Item 125, "Diseases of the urethra, urinary abscesses, etc." is placed with the sex organs rather than with the excretory organs in table 38, because, with very few exceptions, the deaths in this item are sequelae of gonorrhea. Urinary abscesses are secondary usually to urethral stricture,

TABLE 38

Kidneys and related excretory organs

NO.	"CAUSE OF DEATH" AS PER INTERNATIONAL CLASSIFICATION	REGISTRATION AREA U.S.A.		ENGLAND AND WALES 1914	SÃO PAULO 1917
		1906–10	1901–05		
120	Bright's disease	87.4	87.4	37.0	41.2
119	Acute nephritis	10.1	9.6	5.4	29.4
124	Diseases of the bladder	3.1	4.3	3.3	1.3
121, 122	Chyluria and other diseases of the kidneys	2.6	2.8	1.3	9.4
123	Calculi of the urinary passage	0.6	0.5	0.7	0.4
Totals		103.8	104.6	47.7	81.7

which in turn, except for an insignificant number of traumatic cases, is gonorrheal in origin.

Regarding the wisdom of bringing together under one rubric the causes of death listed in table 38 on a biological basis, there would seem to be little doubt.

The "rheumatisms" present another difficult question. A precise and critical decision on the point of where these diseases belong in this present scheme of classification is impossible of attainment. Weighing all the evidence carefully, it seemed best to put chronic rheumatism and gout and acute articular rheumatism in table 39, under "Skeletal and muscular system," rather than here with the kidneys. Much at least of the fatal chronic rheumatism is really a chronic infective arthritis. Gout is a disease due to fundamental disturbances of general metabolism, but the

statistical returns lump deaths from this cause with chronic rheumatism. The death rates from all of these diseases are, fortunately, so small that it makes no essential difference to the final synthetic result towards which we are working where they are placed.

Item 47, "Acute articular rheumatism," and the two tubercular affections, items 32 and 33 (Pott's disease and white swellings) are placed here because the essential lesion produced by the causative agents is in either the bones or the joints.

All of the rates in table 39 are small, and any of the causes of death listed therein could be shifted to other rubrics without sensibly affecting any general result.

TABLE 39

Skeletal and muscular system

NO.	"CAUSE OF DEATH" AS PER INTERNATIONAL CLASSIFICATION	REGISTRATION AREA, U.S.A.		ENGLAND AND WALES 1914	SÃO PAULO 1917
		1906–10	1901–05		
47	Acute articular rheumatism	5.4	5.2	5.6	2.6
146	Diseases of the bones	2.5	2.4	1.5	0.7
48	Chronic rheumatism and gout	2.2	3.6	5.4	0
32	Pott's disease	1.5	1.5	1.6	2.6
33	White swellings	0.7	0.7	0.9	0
147	Diseases of the joints	0.2	0.2	0.4	0
149	Other diseases of the organs of locomotion	0.1	0.1	0.1	0
36	Rickets	*	*	2.7	0.9
Totals		12.6	13.7	18.2	6.8

* Not separately tabulated.

In table 40 are included a number of causes of death beyond those which are included in general heading "V. Diseases of the digestive system" in the International Classification. Of these causes which have been brought in from other parts of the International Classification the first which demands attention is the second on the list "Congenital debility, atrophy, marasmus." This is a part of item 151 of the International Classification. As already pointed out, that item includes "Premature birth," which has in the present classification been placed under "Primary and secondary sex organs" for reasons already stated, and "Congenital debility, atrophy, marasmus, etc.," which is the part included here. The reason for putting this portion of item 151 under the present heading is the practical one that clinical experience shows that the vast majority of the deaths of infants

TABLE 40

Alimentary tract and associated organs concerned in metabolism

NO.	"CAUSE OF DEATH" AS PER INTERNATIONAL CLASSIFICATION	REGISTRATION AREA, – U.S.A.		ENGLAND AND WALES 1914	SÃO PAULO 1917
		1906–10	1901–05		
104	Diarrhea and enteritis (under 2)	96.2	89.0	63.6*	383.6
151†	Congenital debility, atrophy, marasmus	28.8	23.2	27.1	44.3
40	Cancer of the stomach and liver	28.3	24.7	36.5	28.1
1	Typhoid fever	25.6	32.0	4.6	14.4
103	Other diseases of stomach	16.8	17.7	10.9	1.3
105	Diarrhea and enteritis (2 and over)	16.7	20.2		49.9
113	Cirrhosis of the liver	14.3	14.4	11.2	12.2
50	Diabetes	13.7	11.5	12.2	5.4
109	Hernia and intestinal obstruction	12.9	13.0	10.9	3.0
108	Appendicitis and typhlitis	11.2	11.0	7.1	3.5
41	Cancer of the peritoneum, intestines, rectum	8.8	7.1	21.3	4.1
14	Dysentery	6.5	8.6	0.7	9.6
117	Simple peritonitis	6.1	10.8	1.4	13.3
115	Other diseases of the liver	6.1	7.5	2.6	5.7
31	Abdominal tuberculosis	6.0	6.0	9.4	1.7
150†	Other congenital malformations	4.5	3.9	5.4	‡
102	Ulcer of the stomach	3.6	2.9	5.5	3.5
138	Puerperal albuminuria and convulsions	3.4	2.8	1.7	1.7
110	Other diseases of intestines	2.8*	2.9	1.4	5.4
114	Biliary calculi	2.8	2.2	2.3	0.4
39	Cancer of the buccal cavity	2.6	2.1	6.6	1.3
35	Disseminated tuberculosis	2.5	2.8	5.5	2.2
100	Diseases of the pharynx	1.6	1.4	2.1	0.4
13	Cholera nostras	1.0	1.4	0.1	0.2
99	Diseases of the mouth	0.7	0.7	1.5	0.4
59	Other chronic poisonings	0.5	0.5	0	0.2
118	Other diseases of the digestive system	0.5	0.3	0.6	1.1
111	Acute yellow atrophy of liver	0.4	0.4	0.2	0.4
101	Diseases of the esophagus	0.3	0.3	0.2	1.3
57	Chronic lead poisoning	0.2	0.3	0.2	0
26	Pellagra	0.2	0	0	0
49	Scurvy	0.1	0.1	0.1	0.2
106, 107	Parasites	0.1	0.1	0.1	6.3
112	Hydatid tumor of liver	§	§	0.1	0.2
27	Beriberi	¶	¶	0	0.2
12	Asiatic cholera	0	0	0	0
Totals		325.8	321.8	253.1	605.5

* Diarrhea and enteritis, all ages.

† In part.

‡ See footnote to table 35.

§ Death rate less than 0.1 per 100,000.

¶ Not separately tabulated for period named.

which are statistically recorded under this heading "Congenital debility, atrophy, and marasmus" are actually due to deficiencies, functional, structural, or both, in the alimentary tract. In probably more than 95 per cent of all cases "Congenital debility" of an infant means that something is wrong with the alimentary tract in its immediate metabolic functions.

Item 50, "Diabetes," includes deaths from a disease which, while diagnosed from a disarrangement of the excretory function, is primarily an affection of the organs which have to do with the initial or early stages of metabolism (the pancreas, the liver, etc.). It therefore seems to belong properly in the classification where it is now placed rather than with the kidneys. In the International Classification it is included with "General diseases."

The part of item 150 of the International Classification bearing the title "Other congenital malformations" needs some discussion in regard to its inclusion here. In other rubrics of the present classification we have taken account of hydrocephalus and congenital malformation of the heart, both of which come under the general heading "X. Malformations" of the International Classification. The only other rubric under that heading in the International Classification is the one here under discussion "Other congenital malformations." It is, of course, impossible to say in detail what these other congenital malformations are. It seems fair, however, to assume from general knowledge that after hydrocephalus and congenital malformations of the heart are deleted, the great majority of the remaining congenital malformations will relate directly to the alimentary tract or some of its associated organs. Quantitative proof that this is the case is not forthcoming for obvious reasons. The placing of this item here is simply on the basis of the best information it is possible to get from those most familiar with congenital malformations in infants. There is undoubtedly some error inherent in placing this title here, but the net effect of such error must be insignificant for the reason that the death rate under this rubric is very small in total, as will be seen from the table, and furthermore, as has already been stated, it is certain on general grounds that the vast bulk of deaths included here must be due to malformations of the alimentary tract or its associated organs.

Items 31 and 35 (abdominal tuberculosis, and disseminated tuberculosis) are placed here, because, while these titles are somewhat indefinite, it is quite certain that the major portion of the deaths recorded by health officers under these terms are due to tubercular affections of the alimentary tract.

Items 57, 59, 26, 27, and 49 (chronic lead poisoning, other chronic poisonings, pellagra, beriberi, and scurvy) present an interesting problem. The question is whether they should go here or with external causes in table 44. It can be argued that on the one hand, the poisonings are due simply to the ingestion of a deleterious agent and death has no biological basis any more than if a person is struck by an automobile, and, on the other hand, that deaths from the diseases like pellagra and beriberi again simply arise from the fact that the victim lacked a proper diet. But the case is not so simple as this argument would imply. Not all workers in paint factories, nor all inmates of insane asylums or prisons die from these causes. Some survive. And it is reasonable, it seems to me, to suppose that in many cases at least the determining factor in the survival is the relative organic soundness or "strength" of the organs primarily concerned in metabolism. On this basis, this group of causes of death is included in table 40. Fortunately, they are all insignificant contributions to the total death rate.

Item 138, "Puerperal albuminuria," presents a very difficult problem but is included here rather than with other puerperal diseases under the sex organs, or elsewhere, on the reasoning that basically these toxemias are due to faulty maternal metabolism, of unknown origin, which can not in the present state of ignorance be properly charged against any particular organ or organ system. While it is a fact that many women having organically sound excretory organs are able to weather even very severe metabolic storms of this sort near the end of pregnancy and survive, and others with organically weak excretory systems go down, nevertheless it is the general metabolic break-down which is primary. This has led me to change this item from its original position with the kidneys to its present location.

Regarding the other items in table 40, taken from the "General disease" class of the International Classification, there is no need for discussion because it is sufficiently evident that on a biological classification they belong here rather than with any other organ group.

The enormous excess of the São Paulo death rate for the total of the items in table 40 as compared with the Registration Area of the United States and England and Wales is noteworthy. Examination of the data will show that it arises almost entirely from the excessive death rate in São Paulo from diarrhea and enteritis (under 2).

In the main the causes of death included in table 41 in addition to those which appear in class II, "Diseases of the nervous system and of the organs of special sense" of the International Classification, so obviously

belong here as to require no special discussion. Two, however, call for comment. Of these the most important is suicide. In the International Classification suicides are placed under "XIII. External causes," a singularly inept location biologically. The immediate motivation of a suicidal death is surely internal. A searching biological analysis of the phenomenon of suicide has yet to be made, but certain of its biological relations are clear enough. In the broadest terms people commit suicide because their higher cerebral mechanism breaks down under the stresses of the world in which they live, and fails to continue its normal functioning. One of the deepest rooted instincts of the individual among all living things, from lowest to highest, is the instinct for the preservation of the individual life. The only instinct which transcends it, and that only in comparatively few cases in lower animals, is the instinct of reproduction. But the phenomenon of suicide in man marks the complete and total inhibition of this instinct of self-preservation. Suicide is always an act in some degree mentally deliberated before its performance. A constitutionally and hygienically sound mentality weathers the environmental storm which suggests suicide. On the basis of this reasoning suicide death rate is put in table 41.

Item 56, "Alcoholism," is included here because fundamentally deaths so returned would seem to be more truly chargeable against the central nervous system than to any other organ system. This opinion is founded on such results as those of Barrington and Pearson,[4] who conclude, after a careful analysis of data regarding extreme and chronic inebriates, that "there appears for constant age little relation between alcoholism and physical fitness," while between mental defect (and poor education) and alcoholism there is a sensible relation.

> We consider it probable . . . . that the alcoholism is not due to the poor education, nor is it to any marked extent productive of the mental defect, but the want of will-power and self-control associated with the mental defectiveness is itself the antecedent of the poor education and of the alcoholism.

Item 71, "Convulsions of infants," is in the International Classification placed with "Diseases of the nervous system." In the original biological classification it was transferred from that location to table 40 because of the well-known clinical fact that the vast majority of deaths of infants recorded as due to convulsions are really due to profound disarrangements of the alimentary tract, which eventually lead to convulsions. But while

[4] Barrington, A., and Pearson, K., A preliminary study of extreme alcoholism in adults, *Eugenics Lab. Mem.*, XIV, 1910

many infants have profound infections of the alimentary tract, not all have convulsions, and it seems reasonable to suppose that those who do have nervous systems not quite equal to the best in respect of soundness and stability.

TABLE 41

Nervous system and sense organs

NO.	"CAUSE OF DEATH" AS PER INTERNATIONAL CLASSIFICATION	REGISTRATION AREA, U.S.A		ENGLAND AND WALES 1914	SÃO PAULO 1917
		1906–10	1901–05		
61	Meningitis (total)	19.4	31.7	11.5	43.1
66	Paralysis without specified cause	16.1	20.1	7.3	2.6
	Suicide (total)	16.0	13.9	10.0	12.9
71	Convulsions of infants	12.5	21.4	22.7	10.0
30	Tuberculous meningitis	9.1	8.9	12.6	0
56	Alcoholism	5.8	6.1	1.8	2.8
63	Other diseases of the spinal cord	5.8	4.9	7.5	4.4
73 } 74 }	Neuralgia, neuritis and other diseases of the nervous system	5.5	6.9	7.0	2.0
67	General paralysis of the insane	5.5	6.8	6.1	3.0
69	Epilepsy	4.2	4.4	7.6	4.8
68	Other forms of mental alienation	3.6	3.6	2.7	0.7
24	Tetanus	2.7	3.5	0.5	4.6
62	Locomotor ataxia	2.6	2.4	1.9	0.2
76	Diseases of the ears	1.6	1.3	3.3	0
150*	Hydrocephalus	1.4	1.6	1.0	†
60	Encephalitis	1.1	1.9	0.9	1.3
70	Convulsions (non-puerperal)	0.5	1.1	0.3	1.7
72	Chorea	0.2	0.3	0.5	0
23	Rabies	0.2	0.1	0	0.7
75	Diseases of the eye and annexa	0.1	0.1	0.2	0
17	Leprosy	‡	‡	0	5.9
Totals		113.9	141.0	105.4	100.7

* In part.

† See footnote table 35.

‡ Less than 0.1 per 100,000.

The other cause of death needing special comment here is leprosy. I am informed by my friend, Dr. G. H. de Paula Souza, who has had unusual opportunities to know leprosy in all its clinical manifestations, that when this disease becomes fatal it is the nervous system which disintegrates and leads to death.

The first five items in table 42 are affections of the skin about which there can be no doubt respecting the correctness of their inclusion here. The last two items, smallpox and mycoses, are diseases with very low death rates at the present time. Biologically, they represent diseases which either gain entrance through the skin, or in which the principal lesions are of the skin. It therefore appears that on the present scheme of classification they may best be put here.

TABLE 42

The skin

NO.	"CAUSE OF DEATH" AS PER INTERNATIONAL CLASSIFICATION	REGISTRATION AREA. U.S.A.		ENGLAND AND WALES 1914	SÃO PAULO 1917
		1906–10	1901–05		
18	Erysipelas	4.2	4.5	3.1	1.3
44	Cancer of the skin	2.7	2.3	2.6	1.5
144	Acute abscess	1.1	1.4	2.1	0.7
145	Other diseases of the skin	1.0	1.0	3.4	2.4
143	Furuncle	0.5	0.5	0.7	0.2
5	Smallpox	0.2	3.4	0	0.7
25	Mycoses	0.2	*	0.1	1.1
Totals		9.9	13.1	12.0	7.9

* Less than 0.1 per 100,000.

TABLE 43

Endocrinal system

NO.	"CAUSE OF DEATH" AS PER INTERNATIONAL CLASSIFICATION	REGISTRATION AREA, U.S.A.		ENGLAND AND WALES 1914	SÃO PAULO 1917
		1906–10	1901–05		
51	Exophthalmic goiter	1.1	0.7	1.3	0.4
52	Addison's disease	0.4	0.5	0.6	0.7
88	Diseases of the thyroid body	0.4	0.3	0.8	0
Totals		1.9	1.5	2.7	1.1

There is no doubt whatever that the three diseases of table 43 belong biologically with the endocrinal system.

In the foregoing tables have been included all statistically recognized causes of death which it is now possible to classify on an organological basis, and which have a significant death rate. The residue comprises in general three categories (*a*) accidental and homicidal deaths; (*b*) senility; and (*c*) deaths from a variety of causes which are statistically lumped

together and can not be disentangled. Accidental and homicidal deaths find no place in a biological classification of mortality. A man organically sound in every respect may be instantly killed by being struck by a railroad train or an automobile. The best possible case that could be made out for a biological factor in such deaths would be that contributory carelessness or negligence, which is a factor in some portion of accidental deaths, bespeaks a small but definite organic mental inferiority or weakness, and that, therefore, accidental deaths should be charged against the nervous system. This, however, is obviously not sound. For in the first place in many accidents there is no factor of contributory negligence in fact, and in the second place in those cases where such negligence can fairly be alleged its degree or significance is undeterminable and in many cases surely slight.

Senility as a cause of death is not further classifiable on an organological basis. A death really due to old age, in the sense of Metchnikoff, represents, from the point of view of the present discussion, a breaking down or wearing out of all the organ systems of the body contemporaneously. In a strict sense this probably never, or at best extremely rarely, happens. But physicians and registrars of mortality still return a certain number of deaths as due to "senility." Under the circumstances it is not possible to go behind such returns biologically.

The second line of table 44, "Ill-defined diseases," furnishes a striking commentary on the relative efficiency of the medical profession in the United States and England in respect of the reporting of the causes of death. Only about one fourth as many deaths appear in the English vital statistics as due to ill-defined and unknown causes as in the United States figures. Happily, the conditions in this regard are constantly improving in the Registration Area of the United States, due to the well-conceived and untiring efforts of the officials in charge of vital statistics in the Bureau of the Census. They deserve the warmest gratitude of every American vital statistician for the improvements in registration they have brought about.

IV

Having now arranged, so far as possible, all statistically recognized causes of death in a biological classification, we may turn to an examination of the results which such an arrangement shows. In table 45 totals of tables 35 to 44 inclusive, are arranged in descending order of magnitude. The results are shown graphically in figure 24.

TABLE 44

All other causes

NO.	"CAUSE OF DEATH" AS PER INTERNATIONAL CLASSIFICATION	REGISTRATION AREA, U.S.A.		ENGLAND AND WALES 1914	SÃO PAULO 1917
		1906–10	1901–05		
187, 188, 189	All external causes (except suicide)	91.9	87.8	26.1	36.4
	Ill-defined diseases	29.4	47.8	7.3	36.3
154	Senility	29.0	41.0	81.5	11.1
45	Cancer of other organs or of organs not specified	12.9	16.1	16.6	17.9
152*	Other causes peculiar to early infancy	3.4	2.6	5.1	3.3
34	Tuberculosis of other organs	2.1	2.0	1.6	0.2
46	Other tumors (female genital organs excepted)	1.0	1.5	0.5	0.9
55	Other general diseases	1.0	0.5	1.5	3.5
153	Lack of care	0.3	12.3	0.6	0
19	Other epidemic diseases	0.3	0.2	0.6	0.2
Totals		171.3	211.8	141.4	109.8

* In part.

TABLE 45

Showing the relative importance of different organ systems in human mortality

GROUP NO.	ORGAN SYSTEM	DEATH RATES PER 100,000			
		Registration area, U.S.A.		England and Wales 1914	São Paulo 1917
		1906–10	1901–05		
II	Respiratory system	384.9	451.5	395.5	416.0
VI	Alimentary tract and associated organs	325.8	321.8	253.1	605.5
I	Circulatory system, blood	295.0	279.2	302.5	289.9
VII	Nervous system and sense organs	113.9	141.0	105.4	100.7
IV	Kidneys and related excretory organs	103.8	104.6	47.7	81.7
III	Primary and secondary sex organs	88.1	77.4	95.4	103.2
V	Skeletal and muscular system	12.6	13.7	18.2	6.8
VIII	Skin	9.9	13.1	12.0	7.9
IX	Endocrinal system	1.9	1.5	2.7	1.1
	Total death rate classifiable on a biological basis	1335.9	1403.8	1232.5	1612.8
X	All other causes of death	171.3	211.8	141.4	109.8

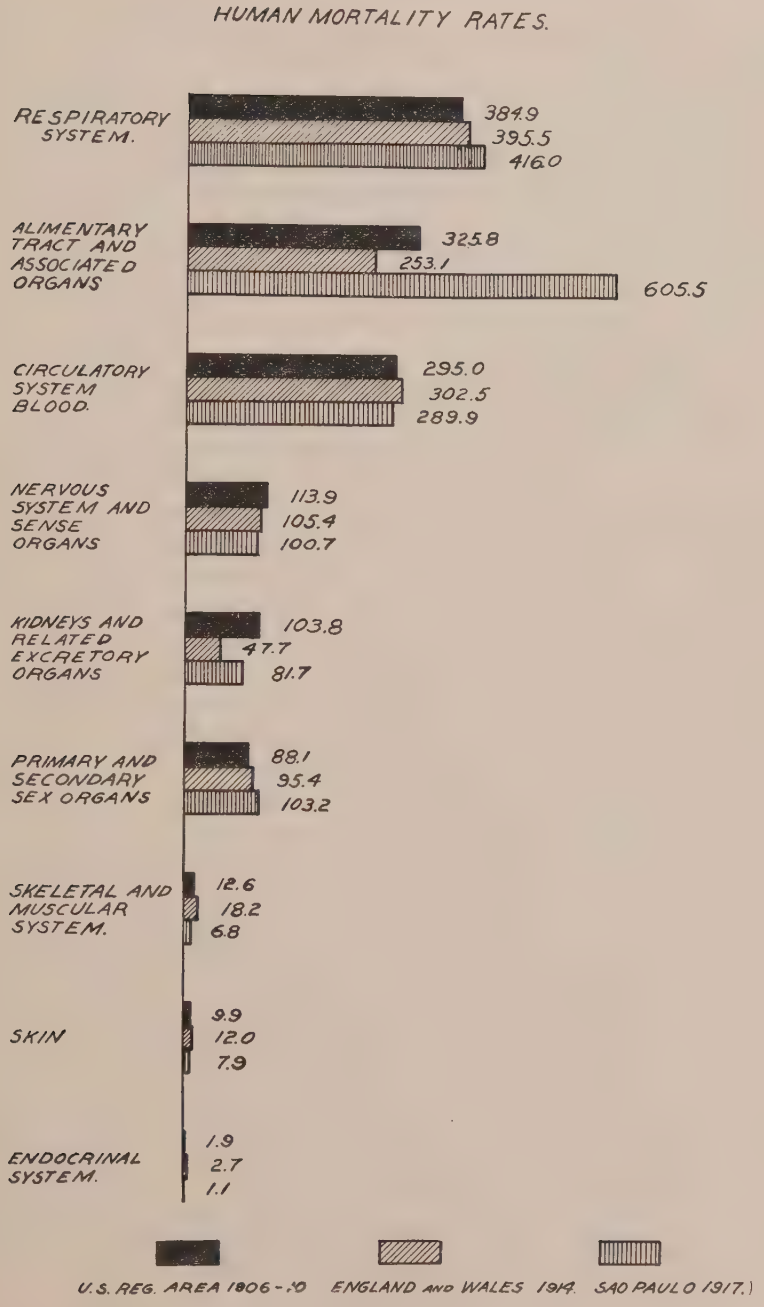


FIG. 24. DIAGRAM SHOWING THE RELATIVE IMPORTANCE OF THE DIFFERENT ORGAN SYSTEMS OF THE BODY IN HUMAN MORTALITY

From table 45 and the diagram a number of noteworthy points can be made out.

1. In the United States, during the decade covered, more deaths resulted from the breakdown of the respiratory system than from the failure of any other organ system of the body. In São Paulo the alimentary tract takes first position, with the respiratory system a fairly close second. The tremendous death rate in São Paulo chargeable to the alimentary tract is chiefly due, as table 40 clearly shows, to the relatively enormous number of deaths of infants under two from diarrhea and enteritis. Nothing approaching such a rate for this category as São Paulo shows is known in this country or England.

2. In all three localities studied the respiratory system and the alimentary tract together account for rather more than half of all the deaths biologically classifiable. These are the two organ systems which, while physically internal, come in contact directly at their surfaces with environmental entities (water, food, and air) with all their bacterial contamination. The only other organ system directly exposed to the environment is the skin. The alimentary canal and the lungs are, of course, in effect invaginated *surfaces* of the body. The mucous membranes which line them are far less resistant to environmental stresses, both physical and chemical, than is the skin with its protecting layers of stratified epithelium.

3. The organs concerned with the blood and its circulation stand third in importance in the mortality list in the United States. Biologically the blood, through its immunological mechanism, constitutes the second line of defense which the body has against noxious invaders. The first line is the resistance of the outer cells of the skin and the lining epithelium of alimentary tract, lungs, and sexual and excretory organs. When invading organisms pass or break down these first two lines of defense the battle is then with the home guard, the cells of the organ systems which, like the industrial workers of a commonwealth, keep the body going as a whole functioning mechanism. Naturally it would be expected that the casualties would be far heavier in the first two defense lines (respiratory and alimentary systems, and blood and circulation) than in the home guard. Death rates when biologically classified bear out this expectation.

4. It is perhaps somewhat surprising that the breakdown of the nervous system is responsible for more deaths than that of the excretory system. But it must be recalled that many deaths in which some degree of breakdown of the excretory system is involved are recorded in official mortality returns under the circulatory system.

5. In the United States the kidneys and related excretory organs are responsible for more deaths than the sex organs. This relation is reversed in England and Wales and in São Paulo. A return to table 37 shows that this difference is mainly due, in the case of England, to two factors, premature birth and cancer. In São Paulo it is due to premature birth and syphilis. Also the difference is partly accounted for by the higher rate from Bright's disease in the United States. The higher premature birth rate for these two localities as compared with the United States might conceivably be explained in either of two ways. It might mean better obstetrics here than in the other localities, or it might mean that the women of this country as a class are somewhat superior physiologically in the matter of reproduction, when they do reproduce. The higher apparent syphilis rate of São Paulo probably means nothing more than better reporting, a less prudish disinclination to report syphilis as a cause of death in São Paulo than in the other two countries.

6. The last three organ systems in the table, skeletal and muscular system, skin and endocrinal organs, are responsible for so few deaths relatively as not to be of serious moment.

7. In a broad sense the efforts of public health and hygiene have been directed against the affections comprised in the first two items in the table, respiratory system and alimentary tract. The figures in the first two columns for the two five-year periods in the United States indicate roughly the rate of progress such measures are making, looking at the matter from a broad biological standpoint. In reference to the respiratory system there was a decline of nearly 15 per cent in the death rate between the two periods. This is substantial. It is practically all accounted for in phthisis, lobar pneumonia and bronchitis. For the alimentary tract the case is not so good—indeed, far worse. Between the two periods the death rate from this cause group did not fall at all! Reference to table 40 shows how all the gain made in typhoid fever was a great deal more than offset by diarrhea and enteritis (under two), congenital debility and cancer. Child welfare, both prenatal and postnatal, seems by long odds the most hopeful direction in which public health activities can expect at the present time substantially to reduce the general death rate. This is a matter fundamentally of education. Ignorant and stupid people must be taught, gently if possible, forcibly if necessary, how to take care of a baby both before and after it is born.

V

We come now to the final stage in this study. Having arranged so far as possible all causes of death on an organological basis, it occurred to me to go one step further back and combine them under the headings of the primary germ layers from which the several organs developed embryologically. To do this is a task of considerable difficulty. It raises intricate, and in some cases still unsettled, questions of embryology. Furthermore the original statistical rubrics under which the data are compiled by registrars of vital statistics were never planned with such an object as this in mind. Still the thing seemed worth trying because of the evolutionary interest which would attach to the result, even though it were somewhat crude and in respect of minor and insignificant details open to captious criticism.

In table 46 the death rates of tables 35 to 43 are subsumed under the three captions, ectoderm, mesoderm and endoderm, according as the organ concerned developed from one or the other of these germ layers. It will be necessary, however, before presenting the tables, to set forth in detail how the figures they contain were made up.

A. Ectoderm. Under this head were placed first, in making up table 46, the totals of table 41 (the nervous system and sense organs), and table 42 (the skin). To the sum obtained by adding these totals together was added (*a*) item 39 (cancer of the buccal cavity) from table 40, on the ground that the lining of the buccal cavity is ectodermal in origin; (*b*) 0.30 of the rates under item 41 (cancer of the peritoneum, intestines and rectum). The point here was that the lining epithelium of the rectum is derived from ectoderm. The cancer rates for these three embryologically different organs, rectum, intestines and peritoneum are arbitrarily lumped together by the registrars of vital statistics. It is necessary for present purposes to unscramble the figures with as little arbitrariness as possible. Data (admittedly rather meager) given by Hoffman[5] (pp. 116–121) from the New York State investigation indicate that in a lumped total of cancer of the peritoneum, intestines and rectum, the fractions incident upon each of the organs are about 0.04 for peritoneum, 0.30 for rectum, and 0.66 for intestines. As these figures are less arbitrary than a mere guess, I have adopted them. It should be remembered that in the final result it makes little difference what fraction is adopted, because the total rate under item 41 is so small. (*c*) Item 86 (diseases of the nasal fossae) is added because the lining membrane of the nose is ectodermal in origin.

[5] Hoffman, F. L., *The Mortality from Cancer Throughout the World*, Newark, 1915.

B. Mesoderm. Here the figures of table 46 were reached by the following process. First, the totals of table 35 (circulatory system), table 37 (sex organs), and table 38 (kidneys), were added together, these being obviously in general mesodermic. From the total so obtained was *subtracted* item 124 of table 38 (diseases of the bladder) since the lining epithelium, the most vulnerable part pathologically, is endodermic in origin. For the same reason item 125 of table 37 (diseases of the urethra) was next subtracted. To the result so obtained was *added* (*a*) the total of table 39 (skeletal system) and item 52 (Addison's disease) from table 43, these representing organs mesodermic in origin; (*b*) 0.04 of the rate under item 41 of table 40 (cancer of peritoneum); (*c*) item 117 (simple peritonitis); (*d*) item 93 (pleurisy). The pleura and peritoneum are mesodermic structures and therefore clearly belong here. The final totals reached after the above described process are those which appear under "Mesoderm" in table 46.

Up to this point in the argument it has been assumed, without discussion, that all the items in table 35 go with the mesoderm. A word may, however, be said about two items. Cerebral hemorrhage and apoplexy (item 64) and softening of the brain (item 65) are brain conditions brought about by a prior functional breakdown of a part of the vascular system, namely the terminal arteries of the brain. Cerebral hemorrhage is due to the rupture of an artery or arteries in the brain, and may in and of itself be a sufficient cause of death, just as would be a hemorrhage due to rupture of an artery in any other part of the body. So far as anything now known can tell us, this fatal accident is as likely as not to occur in a brain of which the nerve cells (of ectodermic origin) are perfectly sound organically. Such a death should be charged against the mesoderm. For suppose it be granted for the moment that there are specific differences between tissues originating from the different germ layers in respect of their likelihood to break down functionally under strain. Then clearly the tendency to any such specificity would be obscured if we charged to ectoderm the breakdown of any organ primarily originating from that germ layer, but where in fact the initial cause of the functional stopping of the proper ectodermic tissue was the prior breakdown of a part of the organ which was mesodermic in origin. This is precisely the condition of affairs relative to the pathology of cerebral hemorrhage.

C. Endoderm. The process of getting the figures here was to add together first the totals of tables 36 and 40 (respiratory system and alimentary tract) the organs represented being mainly endodermal in origin. Then there were *subtracted* from this total the following: (*a*) items 39 (cancer

of the buccal cavity) and 0.34 of item 41 (cancer of the peritoneum, intestines, and rectum), leaving 0.66 of this latter item here for cancer of intestines; (*b*) items 117 (simple peritonitis) and 93 (pleurisy); (*c*) item 86 (diseases of the nasal fossae). All of these items subtracted have been already placed with either ectoderm or mesoderm. Finally, there were *added* items 124 and 125 (diseases of the bladder and of the urethra) which were taken from the mesoderm for reasons already stated under that heading. Also there were added items 51 and 88 from table 43 (exophthalmic goiter and diseases of the thyroid body), because the thyroid arises from the epithelium lining the inner branchial furrows. The result finally obtained by the process described is that which appears in table 46 under "endoderm."

The data of table 46 are shown graphically in percentage form in figure 25.

TABLE 46

Showing the relative influence of the primary germ layers in human mortality

LOCALITY	DEATH RATE PER 100,000 DUE TO FUNCTIONAL BREAKDOWN OF ORGANS EMBRYOLOGICALLY DEVELOPING FROM					
	Ecto-derm	Per cent	Meso-derm	Per cent	Endo-derm	Per cent
U. S. Registration Area, 1906–1910..	129.2	9.7	507.0	38.0	699.7	52.4
U. S. Registration Area, 1901–1905..	158.5	11.3	486.7	34.7	758.6	54.0
England and Wales, 1914.....	130.5	10.6	466.2	37.8	635.8	51.6
São Paulo, 1917.....	111.3	6.9	501.4	31.1	1000.1	62.0

The final results shown in table 46 lead at once to a generalization of considerable interest and significance to the evolutionist. The figures show that in man, the highest product of organic evolution, from 52 to 62 per cent of all the biologically classifiable deaths result from a breakdown and failure further to function of organs arising from the endoderm in their embryological development, while but from 7 to 11 per cent can be regarded as a result of breakdown of organ systems arising from the ectoderm. The remaining 31 to 38 per cent of the mortality results from failure of mesodermic organs. Taking a general view of comparative anatomy and embryology it is evident that in the evolutionary history through which man and the higher vertebrates have passed it is the ectoderm which has been most widely differentiated from its primitive condition, to the validity of which statement the central nervous system furnishes the most eloquent evidence. The endoderm has been least differentiated in the process of evolution, while the mesoderm occupies an intermediate position in this respect. An elaborate array of evidence

might be presented on these points, but to do so would be supererogation. It would amount simply to repeating any standard treatise on the comparative anatomy of the vertebrates.

Degree of differentiation of organs in evolution implies degree of adaptation to environment. The writings of Darwin and Spencer, and in current times of Henry Fairfield Osborn, have demonstrated this point beyond question. From the present point of view we see that that germ layer, the endoderm, which has evolved or become differentiated least in the process of evolution is least able to meet successfully the vicissitudes of the

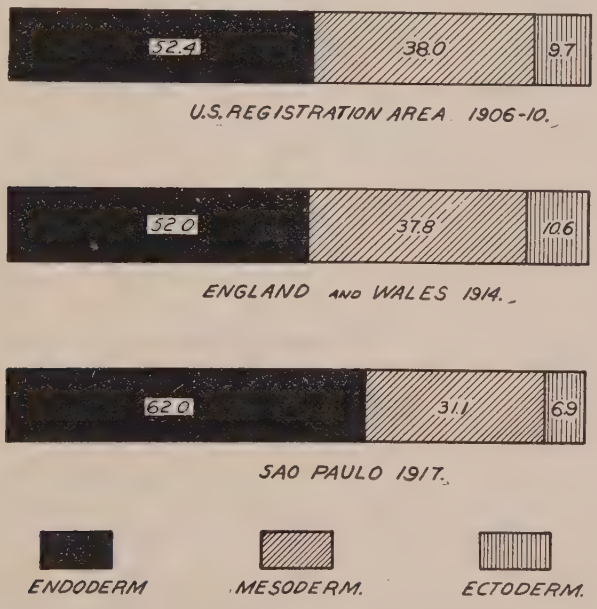


FIG. 25. DIAGRAM SHOWING THE PERCENTAGES OF BIOLOGICALLY CLASSIFIABLE HUMAN MORTALITY RESULTING FROM BREAKDOWN OF ORGANS DEVELOPING FROM THE DIFFERENT GERM LAYERS

environment. The ectoderm has changed most in the course of evolution. The process of differentiation which has produced the central nervous system of man had as a concomitant the differentiation of a protective mechanism, the skull and vertebral column, which very well keeps the delicate and highly organized central nervous system away from direct contact with the environment. The skin exhibits many differentiations of a highly adaptive nature to resist environmental difficulties. It is then not surprising that the organ systems developed from the ectoderm break down and lead to death less frequently than any other.

The figures of table 46 make it clear that man's greatest enemy is his own endoderm. Evolutionally speaking, it is a very old-fashioned and out-of-date ancestral relic, which causes him an infinity of trouble. Practically all public health activities are directed towards overcoming the difficulties which arise because man carries about this antediluvian sort of endoderm. We endeavor to modify the environment, and soften its asperities down to the point where our own inefficient endodermal mechanism can cope with them, by such methods as preventing bacterial contamination of water, food and the like, warming the air we breathe, etc. But our ectoderm requires no such extensive amelioration of the environment. There are at most only a very few if any germs which can gain entrance to the body through the normal, healthy, unbroken skin. We do, to be sure, wear clothes. But it is at least a debatable question whether upon many parts of the earth's surface we should not be better off without them from the point of view of health.

These tables indicate further in another manner how important are the fundamental embryological factors in determining the mortality of man. Of the three localities compared, England and the United States may fairly be regarded as much more advanced in matters of public health and sanitation than São Paulo. This fact is reflected with perfect precision and justice in the relative proportion of the death rates from endoderm and ectoderm. In the United States and England about 52 per cent of the classifiable deaths are chargeable to endoderm and about 10 per cent to ectoderm. In São Paulo 62.0 per cent fall with the endoderm, and but 6.9 per cent with the ectoderm. Since, as we have already shown, public health measures affect practically only the death rate chargeable to endoderm this result which is actually obtained is precisely that which would be expected.

Finally, it seems to me that the results of this study add one more link to the already strong chain of evidence which indicates the highly important part played by innate constitutional biological factors along with environmental factors in the determination of the observed rates of human mortality. Here we have grouped human mortality into broad classes which rest upon a strictly biological basis. When this is done it is found that the proportionate subdivision of the mortality is strikingly similar in such dissimilar environments as the United States, England and Southern Brazil. It is improbable that such congruent results would appear if the environment were the sole or perhaps even the predominant factor in human mortality. This conclusion does not overlook the fact that in some diseases the environment, in a broad sense, is unquestionably the factor of greatest

importance. Nor does it imply that every effort should not be used to measure in every case the precise relative influence of constitution or heredity as compared with environment in the natural history of particular diseases. This constitutes one of the most pressing and difficult problems of medical science.

VI

By way of summary it may be said that the purpose of this study is to rearrange the rates of human mortality as given in official reports of vital statistics, under the code known as the International Classification, into another classification upon a biological basis. The basis taken is organological, each "cause of death" is charged against that organ or organ system, the functional breakdown of which is fundamentally responsible for the death. It is found that from 85 to 90 per cent of all statistically recognized causes of death can be subsumed under such a biological classification. It is found when this is done that the order of significance of the different organ systems in responsibility for human mortality is in general that of the following list, the arrangement being in descending order:

1. Respiratory system
2. Alimentary tract and associated organs
3. Circulatory system and blood
4. Nervous system and sense organs
5. Kidneys and related excretory organs
6. Primary and secondary sex organs
7. Skeletal and muscular system
8. Skin
9. Endocrinal system

The arrangement differs slightly for different countries. If the further step is taken of referring the different organs and organ systems to the primary germ layers from which they embryologically developed, it is found that the death rates chargeable to organs of (*a*) ectodermic, (*b*) mesodermic and (*c*) endodermic origin stand to each other in the case of the United States and England and Wales in the ratio of 1:3.8:5.2 approximately. In the case of São Paulo the ratio is broader, being 1:4.5:9.

VII

The details of the classification followed in the preceding discussion may be set forth in two ways, which together serve as a cross index to all of the 189 causes of death in the Second Decennial Revision of the International List. The biological classification proposes ten main groups as follows:

MAIN HEADS OF BIOLOGICAL CLASSIFICATION OF CAUSES OF HUMAN MORTALITY

All human mortality is due to the breakdown and failure to function of one or another of the bracketed organ groups

- I. Circulatory system, blood, and blood-forming organs.
- II. Respiratory system.
- III. Primary and secondary sex organs.
- IV. Kidneys and related excretory organs.
- V. Skeletal and muscular systems.
- VI. Alimentary tract and associated organs concerned in metabolism.
- VII. Nervous system and sense organs.
- VIII. Skin.
- IX. Endocrinal system.

or to

X. All other causes of death.

In the following list there are given the official International List numbers of the causes of death included in each of the above rubrics of the new classification.

I. Circulatory system, blood and blood-forming organs

Included causes: 2, 3, 4, 6, 7, 11, 15, 16, 20, 21, 22, 53, 54, 64, 65, 77, 78, 79, 80, 81, 82, 83, 84, 85, 116, 142, 150 (congenital malformations of the heart only).

II. Respiratory system

Included causes: 8, 9, 10, 28, 29, 86, 87, 89, 90, 91, 92, 93, 94, 95, 96, 97, 98.

III. Primary and secondary sex organs

Included causes: 37, 38, 42, 43, 125, 126, 127, 128, 129, 130, 131, 132, 133, 134, 135, 136, 137, 139, 140, 141, 151 (premature birth only), 152 (injuries at birth only).

IV. Kidneys and related excretory organs

Included causes: 119, 120, 121, 122, 123, 124.

V. Skeletal and muscular systems

Included causes: 32, 33, 36, 47, 48, 146, 147, 149.

VI. Alimentary tract and associated organs concerned in metabolism

Included causes: 1, 12, 13, 14, 26, 27, 31, 35, 39, 40, 41, 49, 50, 57, 58, 59, 99, 100, 101, 102, 103, 104, 105, 106, 107, 108, 109, 110, 111, 112, 113, 114, 115, 117, 118, 138, 150 (other congenital malformations only), 151 (congenital debility, atrophy, etc. only).

VII. Nervous system and sense organs

Included causes: 17, 23, 24, 30, 56, 60, 61, 62, 63, 66, 67, 68, 69, 70, 71, 72, 73, 74, 75, 76, 150 (hydrocephalus only), 155–163 (all suicides).

VIII. Skin

Included causes: 5, 18, 25, 44, 143, 144, 145.

IX. Endocrinal system

Included causes: 51, 52, 88.

X. All other causes of death

Included causes: 19, 34, 45, 46, 55, 148, 152 (other causes peculiar to early infancy only), 153, 154, 164–186 incl. (all external and accidental causes except suicide), 187–189 incl. (ill-defined diseases, total).

In order to facilitate the finding of the main group in which a particular cause of death of the International List falls the following index will be found useful:

Index to biological classification of causes of death

INTERNATIONAL NUMBER	CAUSE	BIOLOGICAL NUMBER
1	Typhoid fever	VI
2	Typhus fever	I
3	Relapsing fever	I
4	Malaria	I
5	Smallpox	VIII
6	Measles	I
7	Scarlet fever	I
8	Whooping cough	II
9	Diphtheria and croup	II
10	Influenza	II
11	Miliary fever	I
12	Asiatic cholera	VI
13	Cholera nostras	VI
14	Dysentery	VI
15	Plague	I
16	Yellow fever	I
17	Leprosy	VII
18	Erysipelas	VIII
19	Other epidemic diseases	X
20	Purulent infection and septicemia	I
21	Glanders	I
22	Anthrax	I
23	Rabies	VII
24	Tetanus	VII
25	Mycoses	VIII
26	Pellagra	VI
27	Beriberi	VI
28	Tuberculosis of the lungs	II
29	Acute miliary tuberculosis	II
30	Tuberculous meningitis	VII
31	Abdominal tuberculosis	VI
32	Pott's disease	V
33	White swellings	V
34	Tuberculosis of other organs	X
35	Disseminated tuberculosis	VI
36	Rickets	V
37	Syphilis	III
38	Gonococcus infection	III
39	Cancer of the buccal cavity	VI
40	Cancer of the stomach, liver	VI
41	Cancer of the peritoneum, intestines, rectum	VI
42	Cancer of the female genital organs	III
43	Cancer of the breast	III

Index to biological classification of causes of death—Continued

INTERNATIONAL NUMBER	CAUSE	BIOLOGICAL NUMBER
44	Cancer of the skin	VIII
45	Cancer of other organs or of organs not specified	X
46	Other tumors (tumors of the female genital organs excepted)	X
47	Acute articular rheumatism	V
48	Chronic rheumatism and gout	V
49	Scurvy	VI
50	Diabetes	VI
51	Exophthalmic goiter	IX
52	Addison's disease	IX
53	Leukemia	I
54	Anemia, chlorosis	I
55	Other general diseases	X
56	Alcoholism (acute or chronic)	VII
57	Chronic lead poisoning	VI
58	Other chronic occupation poisonings	VI
59	Other chronic poisonings	VI
60	Encephalitis	VII
61	Meningitis	VII
62	Locomotor ataxia	VII
63	Other diseases of the spinal cord (total)	VII
64	Cerebral hemorrage, apoplexy	I
65	Softening of the brain	I
66	Paralysis without specified cause	VII
67	General paralysis of the insane	VII
68	Other forms of mental alienation	VII
69	Epilepsy	VII
70	Convulsions (non-puerperal)	VII
71	Convulsions of infants	VII
72	Chorea	VII
73	Neuralgia and neuritis	VII
74	Other diseases of the nervous system	VII
75	Diseases of the eyes and their annexa	VII
76	Diseases of the ears	VII
77	Pericarditis	I
78	Acute endocarditis	I
79	Organic diseases of the heart	I
80	Angina pectoris	I
81	Diseases of the arteries, atheroma, aneurism, etc.	I
82	Embolism and thrombosis	I
83	Diseases of the veins (varices, hemorrhoids, phlebitis, etc.)	I
84	Diseases of the lymphatic system (lymphangitis, etc.)	I
85	Hemorrhage; other diseases of the circulatory system	I
86	Diseases of the nasal fossae	II

Index to biological classification of causes of death—Continued

INTERNATIONAL NUMBER	CAUSE	BIOLOGICAL NUMBER
87	Diseases of the larynx	II
88	Diseases of the thyroid body	IX
89	Acute bronchitis	II
90	Chronic bronchitis	II
91	Bronchopneumonia	II
92	Pneumonia (total)	II
93	Pleurisy	II
94	Pulmonary congestion, pulmonary apoplexy	II
95	Gangrene of the lung	II
96	Asthma	II
97	Pulmonary emphysema	II
98	Other diseases of the respiratory system (tuberculosis excepted)	II
99	Diseases of the mouth and annexa	VI
100	Diseases of the pharynx	VI
101	Diseases of the esophagus	VI
102	Ulcer of the stomach	VI
103	Other diseases of the stomach (cancer excepted)	VI
104	Diarrhea and enteritis (under 2 years)	VI
105	Diarrhea and enteritis (2 years and over)	VI
106	Ankylostomiasis	VI
107	Intestinal parasites	VI
108	Appendicitis and typhlitis	VI
109	Hernia, intestinal obstruction (total)	VI
110	Other diseases of the intestines	VI
111	Acute yellow atrophy of liver	VI
112	Hydatid tumor of the liver	VI
113	Cirrhosis of the liver	VI
114	Biliary calculi	VI
115	Other diseases of the liver	VI
116	Diseases of the spleen	I
117	Simple peritonitis (non-puerperal)	VI
118	Other diseases of the digestive system (cancer and tuberculosis excepted)	VI
119	Acute nephritis	IV
120	Bright's disease	IV
121	Chyluria	IV
122	Other diseases of the kidneys and annexa	IV
123	Calculi of the urinary passages	IV
124	Diseases of the bladder	IV
125	Diseases of the urethra, urinary abscess, etc	III
126	Diseases of the prostate	III
127	Non-venereal diseases of the male genital organs	III
128	Uterine hemorrhage (non-puerperal)	III

Index to biological classification of causes of death—Concluded

INTERNATIONAL NUMBER	CAUSE	BIOLOGICAL NUMBER
129	Uterine tumor (non-cancerous)	III
130	Other diseases of the uterus	III
131	Cysts and other tumors of the ovary	III
132	Salpingitis and other diseases of the female genital organs	III
133	Non-puerperal diseases of the breast (cancer excepted)	III
134	Accidents of pregnancy	III
135	Puerperal hemorrhage	III
136	Other accidents of labor	III
137	Puerperal septicemia	III
138	Puerperal albuminuria and convulsions	VI
139	Puerperal phlegmasia alba dolens, embolus, sudden death	III
140	Following childbirth (not otherwise defined)	III
141	Puerperal diseases of the breast	III
142	Gangrene	I
143	Furuncle	VIII
144	Acute abscess	VIII
145	Other diseases of the skin and annexa	VIII
146	Diseases of the bones (tuberculosis excepted)	V
147	Diseases of the joints (tuberculosis and rheumatism excepted)	V
148	Amputations	X
149	Other diseases of the organs of locomotion	V
150	Hydrocephalus	VII
150	Congenital malformations of the heart	I
150	Other congenital malformations	VI
151	Congenital debility: premature birth	III
151	Congenital debility: atrophy, marasmus, etc	VI
152	Injuries at birth	III
152	Other causes peculiar to early infancy	X
153	Lack of care	X
154	Senility	X
155 to 163 incl.	Suicide (total)	VII
164 to 186 incl.	All external and accidental causes except suicide	X
187 to 189 incl.	Ill-defined diseases (total)	X

CHAPTER VIII

The Vitality of the Peoples of America[1]

I. THE GENERAL PROBLEM OF RACIAL AMALGAMATION

Science tries ever to peer into the future. Reasoning from the solid basis of human experience in general, that like antecedents are followed by like consequences, it is the constant endeavor of science so to formulate, classify and codify the relations which have in the past subsisted between antecedents and consequences as to be able to pick out the sets which show the maximum degree of constancy, with the thought that then the future may be predicted, if not with absolute certainty, at least with a high degree of probability. Nowhere is the desire to estimate what the future has in store stronger than in the case of matters relating directly to human actions, emotions and thoughts. We know that we are on our way. But where are we going?

This general interest of man in his future social, physical, and mental evolution crystallizes in a specific way for us here and now in the problem of the future of American civilization. That magnificent experiment in human evolution which is called the United States of America has, from its beginning, been at once the embodiment of the loftiest hope and the deep despair of different groups of people. In theory it has always offered what is perhaps the greatest boon intelligent human beings ever crave, equality of opportunity.

The ideal of freedom of opportunity has probably been as consistently maintained in fact, as would in any case be possible, human nature being what it is. Our gates have been open, with some slight and on the whole insignificant exceptions, to all and sundry. Good, bad, and indifferent people have been free to settle here, and have made the fullest use of the freedom. Meantime the country has prospered mightily in material ways, and not insignificantly in things of the spirit, such cheerful pessi-

[1] This material first appeared, under the same title, in the *Amer. Jour. of Hyg.*, vol. 1, pp. 592–674, 1921. It has been altered in the present reprinting only in the direction of elision of parts to avoid repetition of matter appearing elsewhere in this book, and in the correction of typographic and other errors in the original.

This investigation was made, and the paper originally prepared at the request and with the financial aid of the Society of American Peoples, of New York.

mists as Mr. Mencken to the contrary notwithstanding. What vexes the mind is the consideration of the question as to whether the abounding blessings which on the whole have associated themselves with our country have appeared because of, or in spite of, the nearly complete abandon with which all kinds of people have been permitted, indeed encouraged, to join in the enterprise. According to one's views as to the answer to this question he is either fairly satisfied or somewhat disturbed as he looks towards the future.

The essential problem with which the present discussion has to do is that of the racial composition, and biostatistical characteristics in general, of the probable future population of this country. Our judgment on this problem must be determined by a careful examination of what has happened in the past together with what is now going on.

Our problem is purposely stated in this way to emphasize the fact that basically the problem of the future of America is simply one restricted phase of the general problem of population. Whether racial amalgamation or fusion occurs or not, gross population growth goes on steadily. Strange, and indeed inconceivable, as it may appear to many persons, the United States is going to be sometime a densely crowded country, where each individual, consciously or unconsciously, will be challenging the right of every other individual to existence. And, by every present indication, the time when this pressure of population will be definitely felt in this country is not far away. Indeed in terms of the sort of time base by which evolutionary processes are commonly measured, the time of our overcrowding is right upon us. In less than two centuries we may expect a degree of pressure of population upon means of subsistence which will make the everyday life of everybody a different thing entirely from the prodigally wasteful business it now is.

The basis of this time prediction is found in certain mathematical studies which have been made in the writer's laboratory.[2] These studies indicate that, so far as we may rely upon present numerical values, the United States has already passed its period of most rapid population growth, unless there comes into play some factor not now known and which has never operated during the past history of the country to make the rate of growth more rapid. This latter contingency is improbable.

[2] *Cf.* the following papers: Pearl, R., and Reed, L. J., On the rate of growth of the population of the United States since 1790, and its mathematical representation, *Proc. Nat. Acad. Sci.*, vol. 6, pp. 275–288, June, 1920; Pearl, R., and Kelly, F. C., Forecasting the growth of nations, *Harper's Magazine*, vol. 142, pp. 704–713, May, 1921.

The problem of population growth is discussed in detail in Part IV of this book, where the mathematical treatment above referred to is presented.

The probable maximum value of our population is about 197,274,000 roughly. This means that the maximum population of continental United States, as now areally limited, will be roughly twice the present population. This will seem to some a small estimate. It is so easy, and most writers on population have been so prone, to extrapolate population by a geometric series, or by a parabola or some such purely empirical curve, and arrive at stupendous figures, that calm consideration of real probabilities is most difficult to obtain.

The indications are that we shall be uncomfortably close to the asymptotic population by 2100, a time at which the great-grandchildren of a number of persons now living will be components of the population.

What *kind* of a population will that be? What elements now in the population will then, and in the succeeding years, be the dominant ones? Or, will none of the racial groups now so distinct in our population exist as such then, having in the meantime fused and amalgamated, one with the other, till there remains only a biologically homogeneous whole? No one can answer these questions positively today. They are of the future. But we can examine present *tendencies* and past experience. It is to that task that this discussion is addressed. Many others have, of course, written on these problems. Notable papers were those of Mayo-Smith.[3] Others who have written along similar lines are Michaud and Giddings,[4] Giddings,[5] Fishberg,[6] Ripley,[7] and Alleman.[8] But since no recent writer has, so far as I am aware, approached the problem from at all the same point of view as that of the present study, no attempt will be made to review the literature systematically. The point of view throughout this chapter is the formulation of the essential problems on which investigation should be, and profitably could be made, rather than any attempt to solve problems. In such formulations there will, of necessity, be clearly

[3] *Cf.* Mayo-Smith, R., *Emigration and Immigration, a Study in Social Science*, New York, 1890, xiv, 316 pp.; also Immigration and the foreign-born population, *Quart. Publ. Amer. Stat. Assoc.*, vol. 3, pp. 304–320, 1893; also Statistical data for the study of the assimilation of races and nationalities in the United States, *ibid.*, vol. 3, pp. 429–449, 1893; also Theories of mixture of races and nationalities, *Yale Rev.*, vol. 3, no. 2, 1894.

[4] Michaud, G., and Giddings, F. H., The coming race in America, *Century Mag.*, vol. 65, pp. 683–692, March, 1903.

[5] Giddings, F. H., The American people, *Internat. Quarter.*, vol. 7, no. 2, June, 1903.

[6] Fishberg, M., Ethnic factors in immigration, *Proc. Nat. Conf. Char. and Corr.*, 1906, pp. 304–314.

[7] Ripley, W. Z., The European population of the United States, *Jour. Roy. Anthrop. Inst.*, vol. 38, 1908.

[8] Alleman, A., Immigration and the future American race, *Pop. Sci. Monthly*, vol. 5, pp. 586–596, 1909.

pointed out the appalling gaps in our official statistics. Many, indeed most, of the problems to be discussed can only be solved when there is a really adequate system of national bookkeeping, in respect of human life. In pointing out *lacunae* in our national biostatistics the animating motive will be wholly constructive and hopeful of future improvement, and not at all the contrary in respect of either persons or institutions. In general no one is more desirous than the officials charged with the collection and compilation of our national statistics that these statistics should be adequate, in the fullest sense of the word.

II. CRITERIA OF BIOLOGICAL AMERICANIZATION

In a strict sense there are but three general criteria by which the purely biological attributes of a population may be estimated or measured. These are:

1. Somatic physical
2. Somatic psychological
3. Biostatistical

Logically, in a broad sense, all of these may be subsumed under the general title anthropological, but nothing particular is gained by so doing.

In the first category, somatic physical, will be included all those physical attributes of the individuals composing the population, which characterize them as animals, such as hair color, eye color, cephalic index, rate of growth, and so on, right away through the whole gamut of topics commonly designated physical anthropology.

In the second category will be included such attributes as intelligence, habits, feelings, emotions, and such other psychological matters as are capable of definition and measurement with greater or less precision, and at the same time exhibit racial variations of some degree of constancy and specificity.

With neither of these first two criteria shall we deal here, because they have been fully treated by specialists in these particular fields.

The biostatistical criteria form the subject matter of the present discussion. It will be well to examine their scope with some particularity before entering upon detailed treatment of the data.

From the point of view of the population problem, there are two biological phenomena of fundamental importance, *reproduction* and *death*. To study changes in population, of any sort whatever, we need comprehensive and accurate data about each separate element of both of these phenomena. To study progressive or retrogressive secular changes in

population the most useful datum we can have is one which combines within itself the essential variations of both reproduction and death. Such a figure I have called the "vital index" of a population.

We have then the following scheme of essential biostatistical criteria of changes in a people.

	Statistics of	
A. Reproduction	1. Marriages	
	2. Divorces	
	3. Births	*a.* Living
		b. Still
		c. Illegitimate
B. Death	4. Deaths	
C. Vital Index	5. Birth/Death ratio	

Each of the five groups of this scheme have, of course, many subsidiary elements, some of which we shall be able to consider.

There is one general defect of American vital statistics which so constantly will make itself felt in all the discussion which is to follow that it will be well to say what needs to be said about it, once and for all, at the start. The only index of race stock which we have anywhere in our statistics is such as may be inferred from facts as to the nation in which birth occurred. From the point of view of anything approaching exact science this is a most serious defect.

In the first place, in a number of places besides the United States, the population making up the nation includes a number of perfectly distinct racial stocks. Thus a Canadian, in our vital statistics, is just a Canadian, meaning that he was born in the Dominion of Canada, a political entity. In fact he may be an Indian, a Frenchman, an Englishman, an Eskimo, or of any one of a number of other ethnically distinct subdivisions of the human race.

In the second place, there is one highly important racial group, about which we are prevented from getting any biostatistical information. I refer to the Jews. Here, owing to the fact that what is by all odds the biologically purest of all civilized races happens to be also homogeneous in respect of religion, we apparently can have no statistics about it in this country. Whenever an attempt is made, as has repeatedly been the case, to get legislation which will permit the separate statistical enumeration of the Jews, it is blocked by the contention, on the part of some distinguished leaders of that people, that Jewry is a religion and nothing but a religion and does not connote a race. On the other hand others contend that the Jews constitute a highly homogeneous race. Leaving quite aside all discussion of verbal definitions about race and religion, the plain fact cannot

be successfully controverted that the Jews constitute a highly homogeneous group of people in the American commonwealth. It is for this reason and this alone, I take it, that every competent vital statistician agrees as to the great desirability of having statistical information about them. It is greatly to be hoped that in the not distant future this may be accomplished. So far as an entirely dispassionate and unprejudiced observer can see the Jews themselves could lose nothing, and would gain much if this reform could be established.

In the third place no attempt is made in our vital statistics to follow race stocks, even by nation of birth, for more than one generation. In the case of birth statistics we have the birthplace of the propositus and his parents. In the case of death statistics, we have the same information. In the latter case the information obviously extends further back in point of absolute time, but not in point of generations.

The vital statistician in this country has, perforce, to get into an attitude of mind in which he is grateful for what favors are accorded him. Serious as are the *lacunae* which have been mentioned, it is still possible to draw some reasonably accurate conclusions about race stocks in this country. In other words the case is not quite so bad as it might be. The following statement from *Birth Statistics for the Birth Registration Area of the United States*, 1918 (p. 11), "Except for parents born in the United States, however, the country of birth undoubtedly does indicate usually the race stock," unquestionably represents a fact.

We may now turn to the more detailed consideration of the several biostatistical criteria of the changes of populations.

III. MARRIAGE AND DIVORCE

Under our existing conditions of law and custom marriage is a substantially essential prerequisite and concomitant of *normal* human reproduction. This does not mean, of course, that spermatozoa and ova will not successfully unite without benefit of clergy. Too often, alas, they do. But anyone who supposes that *normal* human reproduction goes on in our civilization outside of the marital state should examine the statistics of abortions, stillbirths, congenital malformations, and earliest infant mortality, in the cases respectively of legitimate and illegitimate unions. He will find the conditions in the case of illegitimate unions far indeed from biological normality. Consequently it is essential in the statistical discussion of reproduction to take such a factor into account.

There is furthermore in the present connection an especial reason for discussing marriage statistics. Vast quantities of ink have been spilled in the discussion of the "melting pot" in America. It would be unbelievable if it were not true, in view of the interest in this subject, and its obvious importance as a factor in national well-being, that no data are available by which the degree or extent of the "melting" or racial fusion can be measured on a national scale. What makes the case more curious is the fact that the initial element in the fusion is so easily and directly capable of measurement. The first step in racial fusion in this, as any other country, is obviously inter-racial marriage. But there are in this country absolutely no statistics of national scope about the frequency and character of marriages. In a few states, notably New York and Massachusetts, there is official registration (and compilation) of marriage statistics, by much the same mechanism that registers births and deaths. Drachsler[9] has analyzed the New York marriage figures from the point of view of race amalgamation in a notable memoir. But these are exceptional conditions. To know the extent to which the "melting pot" is actually melting we need as the first datum accurate, uniform and complete federal statistics of marriage on an annual basis, and with racial data included.

As these do not exist now, and there is no immediate hope of getting them, we must resort in the present discussion to indirect methods of learning something of the degree to which racial fusion is occurring at the present time. Happily excellent data are at hand for doing this.

Let us begin by defining *an effective marriage within any specified time duration—say one year—as a marriage which produces living offspring within that time period.* Such marriage unions are reproductively effective within that time. This is, of course, the really important consideration. It could possibly make no effect for good or ill to the country if there were a vast number of say Chinese-American marriages, provided all such unions were reproductively absolutely sterile. They would be biologically non-effective marriages, and would not connote the slightest degree of racial fusion. As has already been pointed out, marriage is the first step in racial fusion, but it is *only* the first step. If the marriage does not become reproductively effective there is no true fusion of the races of the contracting parties. So then what, after all, is needed, in addition to straight marriage statistics, is information as to the racial composition of those marriages which at any particular time are *reproductively effective.*

[9] Drachsler, J., Intermarriage in New York City: A statistical study of the amalgamation of European peoples, *Columbia Univ. Stud. in Hist., Econ., and Pub. Law,* vol. 94, no. 2, pp. 151–356, 1921.

Fortunately data of this latter sort have been regularly published since 1915 for the Birth Registration Area of the United States, collected, checked and compiled by the Division of Vital Statistics of the Bureau of the Census, under the competent direction and supervision of Dr. William H. Davis. Before going into the details as to the character of these statistics it will be well to understand just what is meant by the Birth Registration Area, in order that anyone may judge just what significance is to be attached to its figures, as a sample representing the entire country. In 1919, the year with which we shall deal in the immediately following discussion, the Birth Registration Area had the geographic and demographic characteristics set forth in table 47. This table is modified from *Birth Statistics*, 1919, p. 7.

The states included in the Birth Registration Area in 1919 are shown graphically in figure 26, which is taken from the frontispiece of *Birth Statistics*, 1919.

TABLE 47

Area and population of United States Birth Registration Area in 1919

UNITED STATES		BIRTH REGISTRATION AREA			
Population	Land area (square miles)	Population		Land area	
		Numbers	Per cent of total	Square miles	Per cent of total
104,976,970	2,973,890	61,474,111	58.6	1,075,506	36.2

From these data we see that the area in 1919 included nearly six-tenths of the entire population of the country, and covered nearly four-tenths of the total land area. The Atlantic and Pacific seaboards are practically completely covered, the Middle West is fairly well represented, while the South, the Northwest and the Rocky Mountain regions are practically not represented at all. Probably the most serious omission, from the standpoint of random sampling, is the South. With the reservation which this latter omission implies, we may regard the Birth Registration Area, as constituted in 1919, as fairly representative of the country at large.

In each volume of the *Birth Statistics* issued by the Bureau of the Census is given a table which shows for each child born the birth nationality, in rather wide groups, of both the father and the mother. This table for 1919 is reproduced here in modified form as table 48. The modifications introduced are: (*a*) male and female births, which are given separately in the original, are here combined, since we are not

interested in the sex of the offspring, but only in total marriages effective in 1919; (*b*) the columns and rows of the original table headed respectively "Total foreign," and "Country not stated" are omitted here, as of no purpose in the present inquiry. The net result is that table 48 gives all the definitely known facts regarding birth nationality of the partners *in marriages reproductively effective* in 1919 in the Birth Registration Area.

Table 48 furnishes a picture of the degree of racial assortative mating which was inherent in the marriages which were reproductively effective in 1919 in the Birth Registration Area, or in other words of the degree

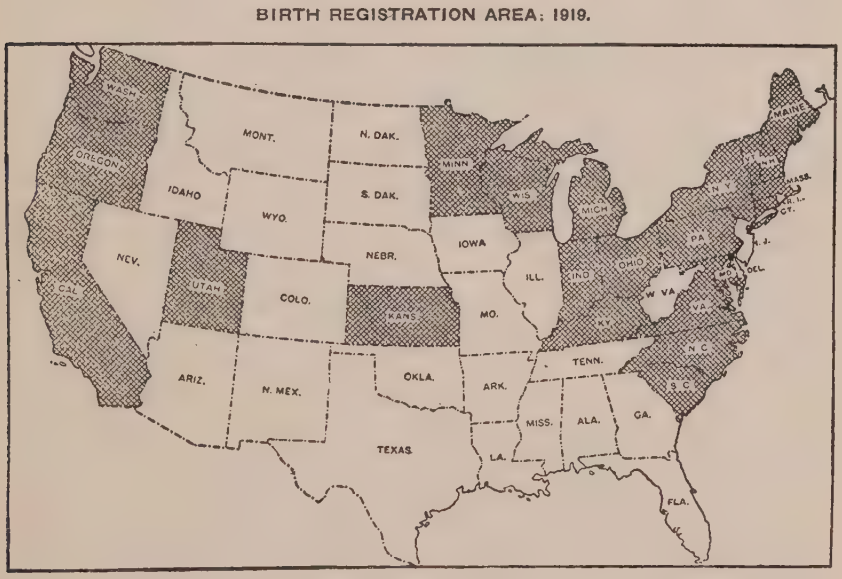


FIG. 26. MAP SHOWING STATES (SHADED) IN THE BIRTH REGISTRATION AREA IN 1919

to which the "melting pot" was melting in that particular year. The data will repay careful and detailed study. One notes at once that the largest frequencies are in those cells which lie on the diagonal of the table connecting the upper left and the lower right corners. These are the cells which exhibit the frequency of effective legitimate matings of like with like in respect of racial stock (in so far as race stock is indicated by nation of birth). Thus there were 816,546 effective marriages in which both male and female were born in the United States: 46,407 in which both members of the pair were born in Austria, and so on. All

TABLE 48

Births (exclusive of stillbirths) of white children, and the country of birth of mother by country of birth of father, in the Registration Area: 1919.

COUNTRY OF BIRTH OF MOTHER	COUNTRY OF BIRTH OF FATHER												TOTALS
	United States	Austria (includes Austrian Poland)	Hungary	Canada	Denmark, Norway and Sweden	England, Scotland and Wales	Ireland	Germany (includes German Poland)	Italy	Poland (not specified)	Russia (includes Russian Poland)	Other foreign countries	
United States	816,546	7,038	1,271	13,033	7,871	8,326	3,788	10,933	11,394	3,634	7,903	8,751	900,488
Austria (includes Austrian Poland)	2,562	46,407	362	34	43	62	22	688	258	615	4,121	599	55,773
Hungary	558	582	14,624	5	8	22	5	307	78	38	313	169	16,709
Canada	11,148	45	7	9,616	111	565	216	121	123	17	65	201	22,235
Denmark, Norway and Sweden	3,583	25	2	110	9,519	140	45	146	32	3	32	222	13,859
England, Scotland and Wales	6,013	92	14	436	143	5,444	459	165	129	23	377	259	13,554
Ireland	5,218	54	4	336	117	702	12,035	140	128	21	29	194	18,978
Germany (includes German Poland)	5,587	465	90	99	132	144	47	6,595	99	216	505	336	14,315
Italy	1,165	65	6	8	8	14	11	11	76,251	8	15	201	77,763
Poland (not specified)	887	274	32	16	11	5	9	97	60	22,562	587	103	24,643
Russia (includes Russian Poland)	2,616	1,670	140	21	25	183	7	310	105	297	53,128	675	59,177
Other foreign countries	3,483	347	73	114	375	204	44	302	503	58	772	30,079	36,354
Totals	859,366	57,064	16,625	23,828	18,363	15,811	16,688	19,815	89,160	27,492	67,847	41,789	1,253,848

other cells of the table except those falling on this diagonal theoretically represent racial mixture or fusion of some sort or other. Actually, as we shall presently see, this theoretical assumption is not entirely justified for all the cells. But neglecting this point for the moment in order first to get a broad view, it is plain enough that any coefficient of assortative mating calculated from the table is bound to give a high value. Persons of the same race tend to mate with each other far more than they tend to go outside the race for a mate. The total number of effective marriages included in the table—over one and a quarter millions—is large enough to be impressive.

The next step obviously is to supplement the general impression derived from mere inspection of the table that there is a high degree of racial associative mating (like mating with like), with a more exact determination of its magnitude. The method which at once occurs to one to do this is to calculate by the contingency method the coefficient of racial assortative mating. This I have not done in the present instance for two reasons: first because I know about what the value would be from experience with other tables of racial assortative mating which I have dealt with by the contingency method before. The second, and more important reason, is that the coefficient in reality tells very little more than common-sense inspection of the table tells, being in fact merely a single numerical expression of that common-sense result, and gives nothing like so much useful information as does the method of analysis of the material upon which we shall presently embark.

Before doing this I wish to discuss at the outstart one source of error implicit in the table which will be neglected throughout, because its magnitude is so small that it cannot sensibly affect any result, whereas to correct for it would require an inordinate expenditure of time and labor, even if, with the available data, it ever could be fully corrected. The error is this. I have referred throughout to the frequencies of table 48 as frequencies of "effective marriages." Actually, of course, they are frequencies of births. Not all births are single births. Some are twins, triplets, etc. In the case of twins the *same* marriage gets counted in table 48 as effective twice in 1919. So, of course, it was in fact from the standpoint of population growth. But from the standpoint of analyzing the racial fusions involved it apparently implies one more pair of parents of the particular sort of racial combinations involved than in fact existed. The number of multiple births, however, in proportion to total births, is so small as to make this a negligible source of error in any conclusions which will here be drawn from the figures. Thus in 1919 there were 12.2 cases of plural

births per 1000 mothers of that year. Or, put in another way, there were only 32,204 live infants born plurally, in a total of 1,373,438 live infants born in 1919.

The first problem on which interest centers may be stated in this way: what proportion of the marriages effective in adding to the population in 1919, were racially pure (race as always being indicated by country of birth), as compared with those which were racially mixed, the mixing being either (*a*) of American-born with foreign-born, or (*b*) of various different sorts of foreign-born together?

To get a first approximation to the answer to this question table 49 has been prepared from table 48 and similar data in *Birth Statistics*, 1919, showing the results for the Registration Area as a whole and for each state separately. The columns contain the following data:

Column A. The sum of the diagonal cells of table 48 (and similar tables for each state). The entries for this column show the total number of births from effective marriages in which both members of the pair were born *in the same country*, and were thus inferentially of the same racial stock. As has already been pointed out this inference is, in a broad way, justified, except for the United States. There we step over into another "universe of discourse." Even there the two members of the married pair are at least alike in these respects, that: (1) they were both born in the United States, and (2) they have been subjected to Americanizing influences for an average period of from twenty-eight to thirty-three[10] years. This means that they went, in general, to American schools and were subjected to Americanizing influences throughout the most formative period of life. Although real racial differences are not to be wiped out in any such easy way or short period as this, still no one who has attentively observed our foreign peoples will deny that the individual born and reared in this country is in many respects definitely different from the foreign-born immigrant, coming at any age after say 12 to 15. For the present then, we shall treat the U. S. × U. S. effective marriages as though they were directly comparable to the other diagonal cell groups; reverting later to further discussion of the point.

Column B. This column contains the sum of the frequencies in the cells forming the first *row* of table 48, *exclusive* of the first cell in the row (U. S. × U. S.). The figure thus shows how many effective marriages in 1919 there were in the B. R. A.,[11] in which the *female* member of the

[10] These figures are respectively about the average ages of mothers and fathers in 1919.

[11] This will be used as a convenient abbreviation of Birth Registration Area throughout the remainder of the discussion.

TABLE 49

Showing for the B. R. A., and each separate state certain relations of effective marriages in 1919

AREA	*A* ALL LIKE × LIKE MATINGS	*B* NATIVE ♀♀ × FOREIGN ♂♂	*C* NATIVE ♂♂ × FOREIGN ♀♀	*D* SUM OF *B* AND *C*	*E* NATIVE ♂♂ × NATIVE ♀♀	*F* ALL FOREIGN PURE MATINGS	*G* ALL FOREIGN MIXED MATINGS	*H* SUM OF *F* AND *G*	*I* PER CENT WHICH *D* IS OF *A*	*J* PER CENT WHICH *D* IS OF *E*	*K* PER CENT WHICH *D* IS OF *F*	*L* PER CENT WHICH *F* IS OF *E*	*M* PER CENT WHICH *G* IS OF *F*	*N* PER CENT WHICH *H* IS OF *E*
Total B. R. A.	1,102,806	83,942	42,820	126,762	816,546	286,260	24,280	310,540	11.49	15.52	44.28	35.06	8.48	38.03
New Hampshire	6,689	996	788	1,784	4,317	2,372	129	2,501	26.67	41.32	75.21	54.95	5.44	57.93
Maine	12,196	1,545	1,448	2,993	9,888	2,308	109	2,417	24.54	30.27	129.68	23.34	4.72	24.44
Massachusetts	67,190	8,217	6,915	15,132	31,468	35,722	2,437	38,159	22.52	48.09	42.36	113.52	6.82	121.26
Minnesota	40,920	6,566	2,625	9,191	33,028	7,892	705	8,597	22.46	27.83	116.46	23.89	8.93	26.03
Washington	19,416	2,455	1,244	3,699	16,412	3,004	458	3,462	19.05	22.54	123.14	18.30	15.25	21.09
Michigan	68,161	7,909	4,080	11,989	50,114	18,047	1,913	19,960	17.59	23.92	66.43	36.01	10.60	39.83
California	42,293	4,888	2,169	7,057	31,333	10,960	1,039	11,999	16.69	22.52	64.39	34.98	9.48	38.30
Wisconsin	45,692	5,277	2,118	7,395	38,329	7,363	678	8,041	16.18	19.29	100.43	19.21	9.21	20.98
Vermont	5,910	551	401	952	4,923	987	63	1,050	16.11	19.34	96.45	20.05	6.38	21.33
Connecticut	27,638	2,663	1,566	4,229	11,468	16,170	1,151	17,321	15.30	36.88	26.15	141.00	7.12	151.04
New York	183,463	17,983	9,202	27,185	93,947	89,516	9,578	99,094	14.82	28.93	30.37	95.28	10.70	105.48
Oregon	11,290	994	513	1,507	10,284	1,006	192	1,198	13.35	14.65	149.80	9.78	19.09	11.65
Utah	11,216	810	611	1,421	10,304	912	103	1,015	12.67	13.79	155.81	8.85	11.29	9.85
Pennsylvania	176,840	13,029	4,789	17,818	117,171	59,669	3,541	63,210	10.08	15.21	29.86	50.92	5.93	53.95
District of Columbia	5,255	300	174	474	4,736	519	66	585	9.02	10.01	91.32	10.96	12.72	12.35
Ohio	99,556	5,067	2,206	7,273	79,342	20,214	1,305	21,519	7.31	9.17	35.98	25.48	6.46	27.12
Maryland	25,347	1,084	535	1,619	22,579	2,768	302	3,070	6.39	7.17	58.49	12.26	10.91	13.60
Kansas	33,180	1,255	530	1,785	31,493	1,687	97	1,784	5.38	5.67	105.81	5.36	5.75	5.66
Indiana	55,027	1,454	566	2,020	51,000	4,027	283	4,310	3.67	3.96	50.16	7.90	7.03	8.45
Virginia	40,334	395	159	554	39,703	631	70	701	1.37	1.40	87.80	1.59	11.09	1.77
Kentucky	52,820	312	112	424	52,532	288	43	331	0.80	0.81	147.22	0.55	14.93	0.63
South Carolina	21,681	84	24	108	21,586	95	10	105	0.50	0.50	113.68	0.44	10.53	0.49
North Carolina	50,692	108	45	153	50,589	103	8	111	0.30	0.30	148.54	0.20	7.77	0.22

pair was American born. This will, in general, indicate the extent of the assimilation of foreign males by the American females of the existing population.

Column C. This contains the sum of the frequencies in the cells forming the first *column* of table 48, again exclusive of the topmost cell (U. S. × U. S.). This is the counterpart of column *B*, showing the extent of effective fusion of American-born male elements with foreign-born females. Columns *B* and *C* together show the whole extent of the effective fusion in the B. R. A. in 1919 of foreign with native-born elements.

Column D gives the sums of the figures in columns *B* and *C* for the purpose above indicated.

Column E gives the total number of effective marriages in which both partners were born in the United States. This is the frequency in the upper left-hand corner cell of table 48, and the corresponding cell for the state tables.

Column F gives the total frequency in all the diagonal cells of table 48, *exclusive* of the corner cells dealt with in column *E*. In other words these figures show the total number of racially *pure* effective marriages in which both partners were foreign-born.

Column G contains the sum of the frequencies in all those cells of table 48 which *are not included* in columns *A*, *B*, and *C* of table 49. These are the cells which fall neither upon the diagonal, nor upon the bordering row or column of the table. The summed frequencies represent the total number of effective marriages in which racial mixture or fusion occurred, with neither partner in the union American-born. The figures show the extent to which the several foreign elements in our population are failing to keep racially pure, without at the same time mixing with American-born.

Column H gives the sums of the entries in columns *F* and *G*. These figures show the total numbers of effective marriages in the B. R. A. in 1919 in which both members of the united pair were of foreign birth.

Column I gives the percentage which each entry in column *D* is of the corresponding entry in column *A*. In other words the figures in column *I* answer the following question: what proportion do the effective marriages which represent a fusion or "melting" of foreign stocks *with American-born elements* bear to the effective marriages in which like mated with like racially (counting U. S. × U. S. as a like mating)?

Column J gives the percentages which the several entries in column *D* are of the corresponding entries in column *E*. These figures show the proportions which effective fusions of native-born with foreign-born ele-

ments bear to effective matings of native-born with native-born. This column may be taken to indicate the rate at which true net amalgamation is taking place.

Column K gives the percentages which the several entries in column *D* are of the corresponding entries in column *F*. The figures show the relative tendencies of foreign stocks to mate (effectively) with native-born stocks, as compared with their tendency to mate (effectively) within their own racial groups in this country.

Column L gives the percentage which each entry in column *F* is of the corresponding entry in column *E*. The figures show the proportions of racially pure effective matings of foreign-born as compared with native-born elements.

Column M gives the percentage which each entry in column *G* is of the corresponding entry in column *F*. The figures indicate the relative tendency of the foreign-born stocks to mate with other foreign-born stocks, as compared with their tendency to mate (among themselves) pure.

Column N gives the percentage which each entry in column *H* is of the corresponding entry in column *E*. The figures compare relatively the effective results of matings in which both parties are foreign-born with those matings in which both partners are native-born.

The several states in table 49 are arranged in descending order of the entries in column *I*.

In the discussion of table 49 it will be well to confine the attention in the first instance to the percentage columns beginning with *I*, noting only that the total number of effective marriages dealt with, over a million, is large enough to command respect. From the table the following points are especially to be noted.

1. For the entire B. R. A. the percentage of amalgamation or fusion of foreign-born stocks with native-born, as compared with racially like effective matings of all sorts, as indicated by effective marriages in 1919, is just under 11.5 per cent. That is, for about every ten effective marriages in which the partners were of like nativity there was one in which one partner was of American and the other of foreign nativity. On the whole this appears a reasonably large proportion, when one considers inherent prejudices which must be overcome before such marriages can occur. Of course one realizes fully that some unknown, but certainly significant proportion of the 126,762 American × foreign-born cross matings are really not *racially* cross at all but like, one partner merely having been born in the United States. But fully granting this, there are two further points to be noted, namely, first, that American birth and upbringing to

marrying age tend to create a considerable measure of antipathy or prejudice towards the more recent immigrant of the same race. In making this statement the writer is relying on his own personal observations of foreign stocks in our country. In many cases the American-born child of foreign parents desires passionately to be a "pure American," to slough off and forget the attributes which characterize, as he or she thinks, the "wop," using this as a generic term of disparagement for the foreigner. Un-American language, names and customs of all sorts, excite in many cases a positively phobic reaction in the native-born children of foreign-born parents. How unfortunate this all is is only too often apparent. The American substitutes for the dropped foreign habits are frequently poor substitutes indeed. The ignorant and foolish child assimilates the worst of American habits and manners, while letting go of things often distinctly fine and precious.

In the second place it must be remembered that in the 816,546 effective marriages of native-born there are many racially unlike matings. Perhaps this balances off in good part the error inherent in cross-mating figures.

2. Thirteen states exhibit a fusion percentage higher than the average, with New Hampshire followed by Maine at the top, with approximately one-fifth of all the effective marriages between native- and foreign-born partners. In these two states the vast majority of these mixed marriages includes a French Canadian as one of the partners. The group above the average in fusion percentage includes all the New England States except Rhode Island (which is not in the B. R. A.), and New York. This would be reasonably expected. These are old states in which (*a*) there has been time for the more superficial and frothy aspects of race prejudice to evaporate, and (*b*) the foreign population has, by and large, been of rather distinctly superior quality. In the Middle West, Minnesota, Michigan, and Wisconsin give percentages well above the average. Here again superior foreign-born elements have been long in residence and demonstrated their worth. From the Far West, Washington, California, Oregon and Utah give percentages more or less above the average. Perhaps this is to be accounted for by the rather distinctly open-minded racial tolerance of these communities, so long as Orientals are not involved.

3. The distinctive feature of the group of states giving fusion percentages below the average is that it is outstandingly a southern group. Six out of the ten states in the group lie south of the Mason-Dixon line. This is probably to be accounted for by two factors: (*a*) an intense prejudice against racial miscegenation in general, founded upon the concrete case

of the negro, and (*b*) a relatively small proportion of foreign-born in the communities concerned.

4. This brings up a question of obvious interest. When the states are arranged in descending order of effective race fusion percentages as in table 49 (from column *I*), is not this also about the same order in which they would stand in respect of percentage of foreign-born white popula-

TABLE 50

Comparing percentages of foreign-born whites in the total population in 1910 with Column I of Table 49

STATES	COLUMN *I*, TABLE 49 FUSION PERCENTAGE	PER CENT OF FOREIGN-BORN WHITES IN POPULATION IN 1910
New Hampshire	26.67	22.4
Maine	24.54	14.8
Massachusetts	22.52	31.2
Minnesota	22.46	26.2
Washington	19.05	21.1
Michigan	17.59	21.2
California	16.69	21.8
Wisconsin	16.18	22.0
Vermont	16.11	14.0
Connecticut	15.30	29.5
New York	14.82	29.9
Oregon	13.35	15.3
Utah	12.67	17.0
Pennsylvania	10.08	18.8
District of Columbia	9.02	7.4
Ohio	7.31	12.5
Maryland	6.39	8.0
Kansas	5.38	8.0
Indiana	3.67	5.9
Virginia	1.37	1.3
Kentucky	0.80	1.7
South Carolina	0.50	0.4
North Carolina	0.30	0.3

tions? Table 50 shows that this is in fact the case to a high degree of approximation.

How close the parallelism is between the two columns of table 50 is shown graphically in figure 27.

The important fact strikingly demonstrated in table 50 and figure 27 is that the amount of racial amalgamation or fusion going on in the several parts of the American "melting pot" is proportional in the most direct

and close way to the amount of foreign-born white stock in the local population. In other words the dominant and outstanding factor in determining whether there shall be true effective assimilation of foreign elements into the established American population is simply the opportunity afforded by propinquity—which is statistically one of the most potent factors in bringing about any and all marriages. Given a community in which there are many foreign-born whites as more or less settled resi-

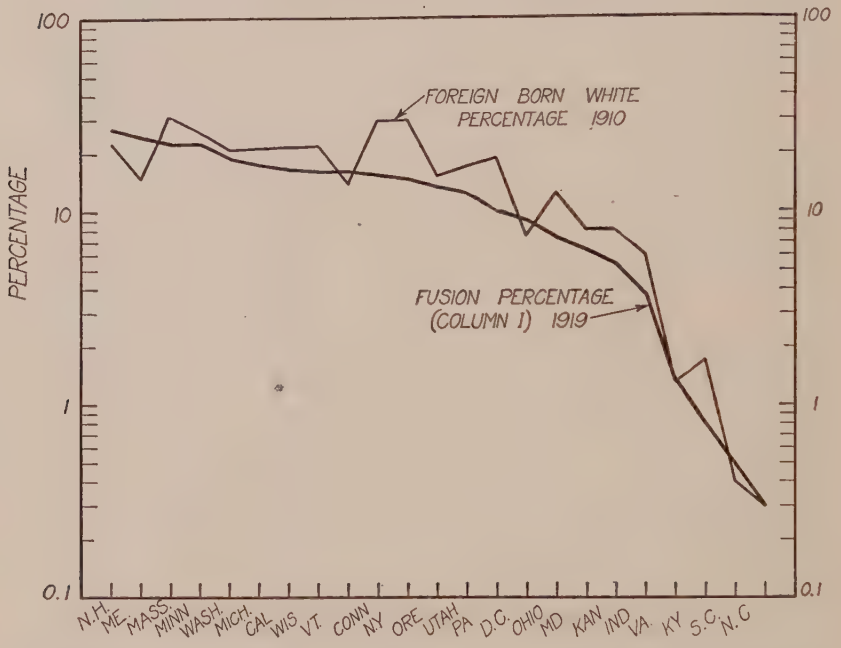


FIG. 27. SHOWING THE CLOSE AGREEMENT WHICH EXISTS BETWEEN THE PERCENTAGES OF MIXED MARRIAGES OF NATIVE AND FOREIGN-BORN EFFECTIVE IN 1919, AND THE PERCENTAGES IN THE POPULATION OF FOREIGN-BORN WHITES IN 1910

dents, there will be relatively many marriages in which one of the partners will be of American and the other of foreign nativity.

5. Turning to column *J* of table 49, which gives the percentage which the total number of reproductively effective cross-matings, in which one partner was American-born, is of the number of effective matings in which both partners are native-born, it is seen that while the percentages are, as must necessarily be the case, somewhat higher than those of column *I*, they nevertheless follow about the same order. Some of the individual

entries are of interest. Thus in the case of Massachusetts there were nearly one-half (48.09 per cent) as many matings effective in producing offspring in 1919 in which fusion of American- and foreign-born occurred, as there were effective matings where both partners were native-born. Just as in the preceding case it is evident that opportunity is the important factor in making the melting pot melt. Figure 28 is of the same general character as figure 27 except that here the *J* column percentages, which may be taken as the closest approximation to actual, net, effective

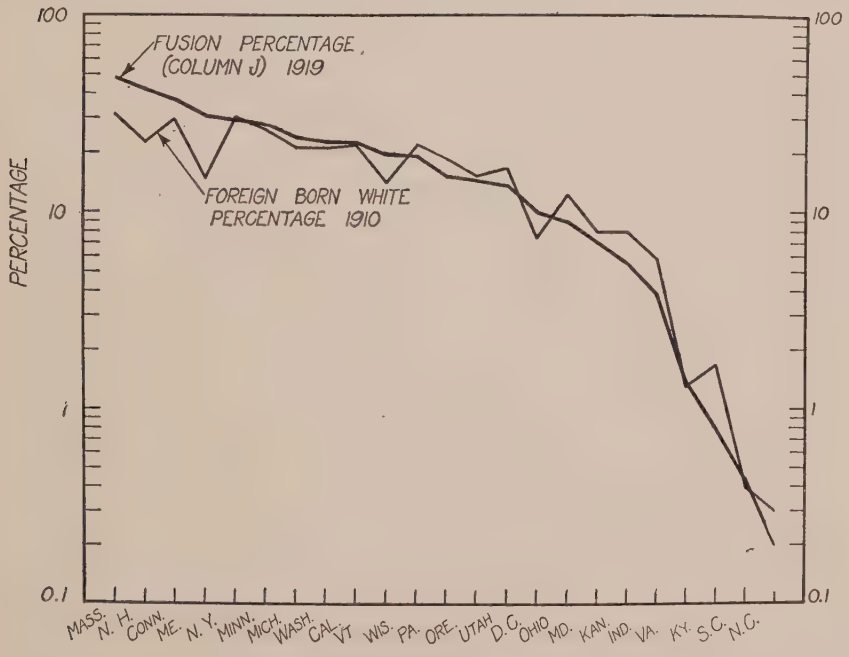

100
10
1
0.1
PERCENTAGE
FUSION PERCENTAGE (COLUMN J) 1919
FOREIGN BORN WHITE PERCENTAGE 1910
MASS. N. H. CONN. ME. N. Y. MINN. MICH. WASH. CAL. VT. WIS. PA. ORE. UTAH D. C. OHIO MD. KAN. IND. VA. KY. S. C. N. C.

FIG. 28. SHOWING THE AGREEMENT BETWEEN PERCENTAGES OF MIXED AMERICAN AND FOREIGN EFFECTIVE MARRIAGES IN 1919 (PERCENTAGES ON NATIVE × NATIVE BASE ONLY), AND PERCENTAGES OF FOREIGN-BORN WHITES IN THE POPULATION IN 1910

racial amalgamation, are plotted against percentages of foreign-born whites in 1910.

The agreement between the two lines is even closer in this diagram than was the case in figure 27. There can be no doubt that where the most foreign-born whites are, there occurs the most mixed mating with native-born, not only absolutely but also relatively, in proportion both to all racially pure matings and to purely American (*i.e.*, native × native) matings.

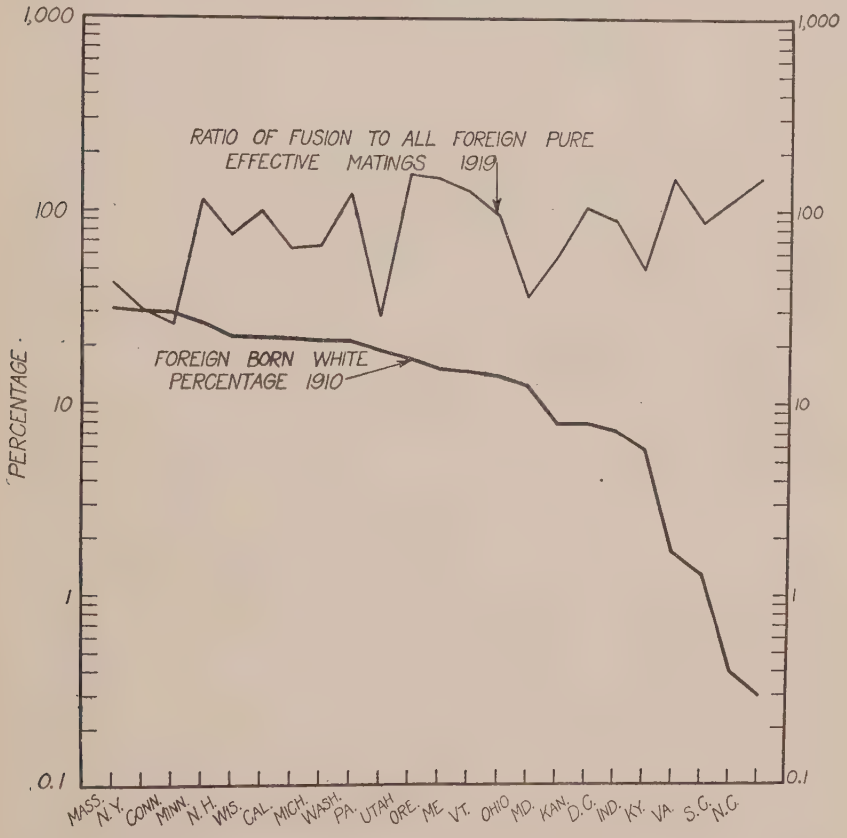
And there can be no doubt, under the circumstances, about the reality of racial fusion in this country. When from one-fourth to one-half as many children are produced in a given year from mixed matings (in which one partner is American) as from matings of strictly American-born persons, amalgamation of the immigrant elements into the previously existing complex is certainly going on apace.

6. Column *K* gives us a view of the same phenomenon from another angle. Here the question presented is: What proportion do effective mixed matings (in which one partner is American-born) bear to racially pure matings in the same communities in which both partners are of foreign birth? In ten out of the twenty-three states there are *more* effective mixed matings of the amalgamating type—or, if one chooses, of the Americanizing type—than there are of racially pure foreign × foreign matings. These are the states exhibiting percentages greater than 100 in column *K*. A better appreciation of the essential point here can be gained by a graphic presentation. This is given in figure 29, where the data of column *K* are plotted against the percentages of foreign-born whites in 1910. In the diagram the states are arranged in descending order of proportion of foreign-born whites in 1910 (heavy line). This, it will be noticed, is the opposite of the plan in figures 27 and 28, where these data were given as the light instead of the heavy line.

It is at once apparent that, in general, there is no significant association between the two variables here plotted. Leaving out of account for the moment the three states with the highest percentage of foreign-born whites, namely, Massachusetts, New York and Connecticut, it is clear that the remaining twenty states proceed across the diagram in what amounts to a horizontal line, in respect of the proportion of fusion to pure all-foreign matings, while at the same time the percentage of foreign-born in the population is steadily decreasing. In the eastern states mentioned the case is different. The proportion of foreign-born whites in total population is there highest. The proportion of fusion matings to pure foreign-born matings is practically the lowest. Here we see the effect of the condensed, homogeneous "foreign colony" type of population, flourishing in great industrial centers. Yet the fact must not be overlooked that all three of these states, Massachusetts, Connecticut and New York, are above the average in the percentage which fusion matings form of pure American-born matings (*cf.* column *J*). There is no contradiction in these two facts, though at first glance there might appear to be. The facts merely mean that in each of these three commonwealths there is a comparatively large, well-settled, foreign-born population, reproducing at a

relatively much more rapid rate, both pure and when fused with American-born stock, than is the strictly American-born stock by itself.

7. Column L gives for each state the percentage which effective pure foreign-born $\times$ foreign-born matings form of the effective native-born $\times$


1,000
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PERCENTAGE
RATIO OF FUSION TO ALL FOREIGN PURE EFFECTIVE MATINGS 1919
FOREIGN BORN WHITE PERCENTAGE 1910
MASS. N.Y. CONN. MINN. N.H. WIS. CAL. MICH. WASH. PA. UTAH ORE. ME. VT. OHIO MD. KAN. D.C. IND. KY. VA. S.C. N.C.

FIG. 29. SHOWING THE RELATION BETWEEN (*a*) THE PROPORTION OF FOREIGN-BORN WHITES IN THE POPULATION IN 1910, AND (*b*) THE PERCENTAGE WHICH "FUSION" EFFECTIVE MATINGS (AS DEFINED IN TEXT) ARE OF EFFECTIVE ALL-FOREIGN PURE MATINGS IN THE SAME COMMUNITIES

native-born matings. Plotting these results against the percentage of foreign-born in the total population in 1910 gives the result shown in figure 30.

It is evident that the two curves run generally together, meaning that the proportion of foreign $\times$ foreign effective pure matings tends to be

highest where there are proportionally the greatest number of foreign-born in the population, and lowest where there are the fewest. But there are some striking outstanding points, which merit special consideration. In the three states Massachusetts, New York and Connecticut there were

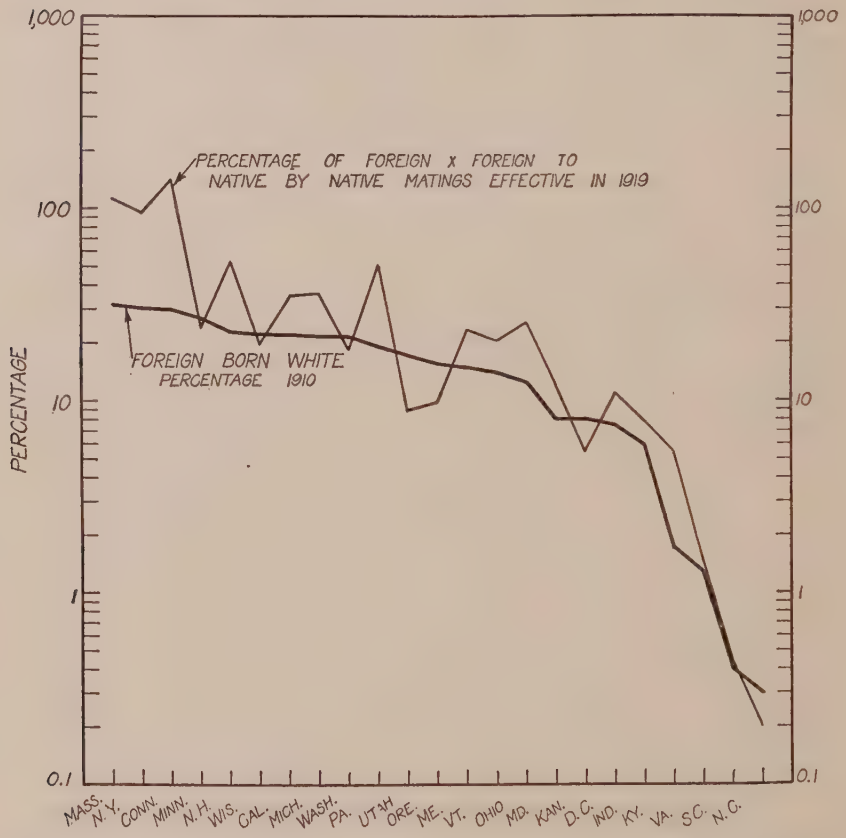

FIG. 30. SHOWING THE RELATION BETWEEN (*a*) PERCENTAGE OF FOREIGN-BORN IN TOTAL POPULATION IN 1910 (HEAVY LINE), AND (*b*) PROPORTION OF FOREIGN × FOREIGN MATINGS TO NATIVE MATINGS EFFECTIVE IN 1919

actually, in 1919, nearly as many or more matings of the type pure foreign-born × foreign-born as there were of the type native-born × native-born, *effective in producing offspring*. This means, put in another way, that in these three states substantially as large or larger additions to the future population were made in 1919 by the foreign-born as by the

native-born. Furthermore in two other states, namely New Hampshire and Pennsylvania the effective pure foreign × foreign matings were more than half as numerous as the effective native × native.

There can be no doubt that, on the average, by whatsoever standard measured, the native-born are "Americanized" in higher degree than the foreign-born. It would appear that in the five states named, the less Americanized elements are multiplying too fast in proportion to the more Americanized. There can furthermore be no doubt that this state of affairs arises in largest degree as a consequence of the crowding of large, purely foreign "colonies" in great industrial centers of population. This is certainly true of Massachusetts, New York, Connecticut and Pennsylvania. In New Hampshire the same mechanism essentially is at work and is the *causa princeps* of the result, but it operates chiefly on a different sort of foreigner, namely the French-Canadian, about whose eventual and satisfactory Americanization no particular doubt can be felt. In the other four states named, what appears to be needed chiefly is to break up the too close geographical segregation. Then the problem will take care of itself. This appears to me to be abundantly proved by the results which have already been presented here. Give the foreign elements a chance to meet and really know the native population, and the melting pot promptly begins to seethe and bubble. The blame for the sort of conditions depicted by the statistics of Massachusetts, New York, Connecticut and Pennsylvania, in my judgment, lies far heavier upon the native Americans than upon the foreigners. Similarly situated we should all act much as the foreigners do in respect of our mating habits.

8. Column *M* presents another angle of the problem. It shows the percentage which effective all-foreign mixed matings are of effective all-foreign pure matings. The all-foreign mixed matings are those in which both partners are foreign-born, but the husband has a different country of birth than the wife. In other words we have in this column a measure of the extent to which assortative mating in respect of race occurs among the foreign-born in this country, excluding all mixtures of foreign with American-born. It is seen at once that all percentages are relatively small, the largest being 19.09 in the case of Oregon. The comparison between the entries of column *M* and those of column *K* is highly significant. The foreigner in this country is much more likely to marry an American-born person, if he does not marry one of his own race, than he is to marry some other foreigner not of his own race. This fact would become even more emphasized if we were able to make the proper corrections for Jews of different countries of birth.

9. Column *N* compares the total of effective matings of foreign × foreign-born (whether pure or mixed) with the total of native × native effective matings. In three states, Massachusetts, New York and Connecticut, these percentages are over 100, as would of course be expected from what has gone before. In these states the foreign-born produced more children in 1919 than did the native-born. But, after all, the *children* were native-born and will be subjected to Americanizing influences from birth onwards.

Up to this point we have been considering very broad types of matings, and examining the extent of effective fusion of races in different parts of the B. R. A. It will now be well to examine the facts from another angle, and from one point of view summarize them.

Table 51 shows that in 1919 there were in the B. R. A. 78 racially different types of mating effective in producing offspring, not separating sexes of either mated partners or offspring. These matings are given in descending order of number of children produced. It is to be understood, here as before, of course, that "race combination" merely means "so far as indicated by country of birth," and is in so far and in that sense not accurate.

From table 51 it is seen that 65 per cent of the children produced in the B. R. A. in 1919 were from American-born parents. Of the remaining 35 per cent the make-up was as follows: 10.113 per cent of the total children had one parent native-born, leaving approximately 25 per cent of the total births having both parents foreign-born. So then it appears that almost exactly 75 per cent of the total children had one or both parents native-born. Of the foreign-born effective matings the highest percentage is exhibited by the Italians, next by the Russian born, next by the Austrian born, and next by the residue group "other foreigners." These four foreign groups contributed together some 16 per cent of the total children. It is probably conservative to estimate that one-half at least of these parental groups were Jewish. The first twelve of the matings in the table produced over 90 per cent of the children born in the B. R. A. in 1919. Whether the above facts, which are shown graphically in figure 31, will be regarded as alarming or reassuring depends upon one's preconceived point of view. From one point of view it will be regarded an evil of alarming proportions that but 65 per cent of children born in this country have both parents native-born. But, on the other hand, as the figures show, the proportion having both parents native-born is overwhelmingly larger than that for any other mating. Our present task, however, is simply to present the facts, to as great a degree of approximation as is possible.

TABLE 51

Net effectiveness in producing next generation

ORDER	RACE COMBINATION	NUMBER OF BIRTHS	PER CENT
1	United States × United States	816,546	65.123
2	Italy × Italy	76,251	6.081
3	Russia × Russia	53,128	4.237
4	Austria × Austria	46,407	3.701
5	Other foreign × other foreign	30,079	2.399
6	United States × Canada	24,181	1.929
7	Poland × Poland	22,562	1.799
8	United States × Germany	16,520	1.318
9	Hungary × Hungary	14,624	1.166
10	United States × United Kingdom	14,339	1.144
11	United States × Italy	12,559	1.002
12	United States × other foreign	12,234	0.976
13	Ireland × Ireland	12,035	0.960
14	United States × Scandinavia	11,454	0.914
15	United States × Russia	10,519	0.839
16	Canada × Canada	9,616	0.767
17	United States × Austria	9,600	0.766
18	Scandinavia × Scandinavia	9,519	0.759
19	United States × Ireland	9,006	0.718
20	Germany × Germany	6,595	0.526
21	Austria × Russia	5,791	0.462
22	United Kingdom × United Kingdom	5,444	0.434
23	United States × Poland	4,521	0.361
24	United States × Hungary	1,829	0.146
25	Russia × other foreign	1,447	0.115
26	United Kingdom × Ireland	1,161	0.093
27	Austria × Germany	1,153	0.092
28	Canada × United Kingdom	1,001	0.080
29	Austria × other foreign	946	0.075
30	Austria × Hungary	944	0.075
31	Austria × Poland	889	0.071
32	Poland × Russia	884	0.071
33	Germany × Russia	815	0.065
34	Italy × other foreign	704	0.056
35	Germany × other foreign	638	0.051
36	Scandinavia × other foreign	597	0.048
37	United Kingdom × Russia	560	0.045
38	Canada × Ireland	552	0.044
39	United Kingdom × other foreign	463	0.037
40	Hungary × Russia	453	0.036
41	Hungary × Germany	397	0.032
42	Austria × Italy	323	0.026

TABLE 51—*Continued*

ORDER	RACE COMBINATION	NUMBER OF BIRTHS	PER CENT
43	Canada × other foreign	315	0.025
44	Germany × Poland	313	0.025
45	United Kingdom × Germany	309	0.025
46	Scandinavia × United Kingdom	283	0.023
47	Scandinavia × Germany	278	0.022
48	Hungary × other foreign	242	0.019
49	Ireland × other foreign	238	0.019
50	Canada × Scandinavia	221	0.018
51	Canada × Germany	220	0.018
52	Ireland × Germany	187	0.015
53	Scandinavia × Ireland	162	0.013
54	Poland × other foreign	161	0.013
55	Austria × United Kingdom	154	0.012
56	United Kingdom × Italy	143	0.011
57	Ireland × Italy	139	0.011
58	Canada × Italy	131	0.010
59	Italy × Russia	120	0.0096
60	Germany × Italy	110	0.0088
61	Canada × Russia	86	0.0069
62	Hungary × Italy	84	0.0067
63	Austria × Canada	79	0.0063
64	Austria × Ireland	76	0.0061
65	Hungary × Poland	70	0.0056
66	Austria × Scandinavia	68	0.0054
67	Italy × Poland	68	0.0054
68	Scandinavia × Russia	57	0.0045
69	Scandinavia × Italy	40	0.0032
70	Hungary × United Kingdom	36	0.0029
71	Ireland × Russia	36	0.0029
72	Canada × Poland	33	0.0026
73	Ireland × Poland	30	0.0024
74	United Kingdom × Poland	28	0.0022
75	Scandinavia × Poland	14	0.0011
76	Hungary × Canada	12	0.00096
77	Hungary × Scandinavia	10	0.0008
78	Hungary × Ireland	9	0.0007
Total		1,253,848	100.003

Throughout the discussion of this point we have been compelled, by lack of the raw statistical data necessary to do anything else, to take country of birth as indicative of race. Everyone knows that it is not accurately so. It is at best only an approximation, and conceals many

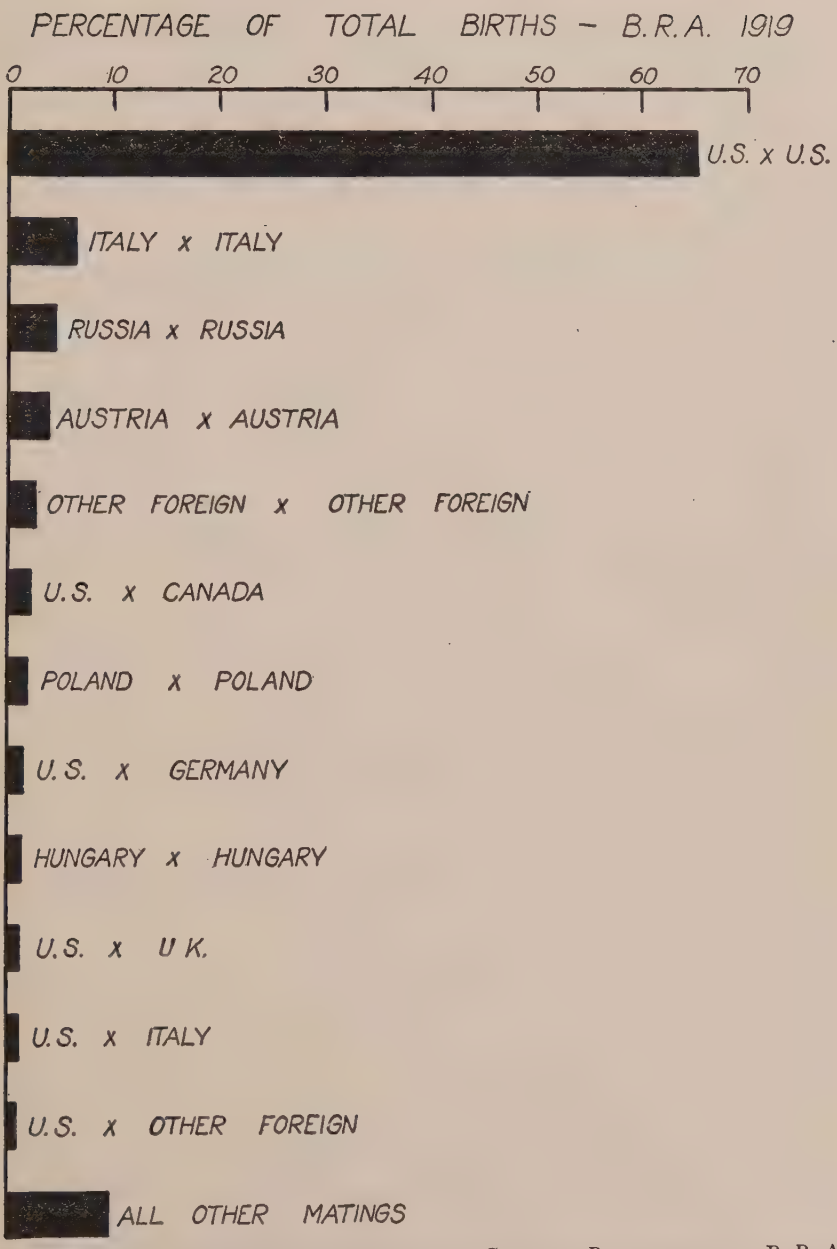

FIG. 31. BAR DIAGRAM SHOWING PROPORTION OF CHILDREN PRODUCED IN THE B. R. A. IN 1919, FROM DIFFERENT TYPES OF MATINGS

pertinent facts. Again, because of the lack of the necessary data, it is impossible to make any correction of tables 48, 49 and 51 which will, after the expenditure of a considerable amount of time that would be involved in attempting corrections, tell us anything more exact than what we know to be the broad features of the case. The errors in these tables incident to taking birthplace as an index of race are, broadly speaking, as follows:

1. An error which probably tends to make the degree of racial assortative mating in effective marriages appear *lower* than it really is, is found in the fact that the U. S. × U. S. group contains many different racial elements. Many of these probably mate assortatively and should, on a strictly racial basis, be taken out of the upper left hand corner cell of table 48 and distributed to their proper diagonal cells down the table. But others of this sort do not mate in strict racial assorting, and they should be distributed to the appropriate cells of the table off the diagonal.

2. Another factor of much greater importance in lowering the apparent below the true degree of racial assortative mating, is found in the cells not on the diagonal in table 48 with disproportionately high frequencies; as for example, U. S. ♂ × Russian ♀ (frequency 2616), Austrian ♂ × Russian ♀ (frequency 1670), etc. Now while these appear to be cross-matings, as a matter of obvious fact, they are in the vast majority of cases strictly pure matings, namely of Jewish ♂ × Jewish ♀. They should in some unknown, but certainly large proportion, be transferred to the diagonal cells. Another example of the same thing, but not involving Jews, is seen in the disproportionately high frequencies in the cells Canadian ♂ × U. K. ♀ (frequency 436) and U. K. ♂ × Canada ♀ (frequency 565). What we really have, in a great proportion of these cases, is the mating of Canadian-born English, Welsh or Irish males with foreign-born females *of the same race.*

The first of the sources of error named I am personally inclined largely to discount, because of the conviction that about the only logically tenable simple definition of an American, in the sense the word is here used, is that of nativity. An American is a person born in the United States.[12] The only *racially* pure American is the Amerind. Everybody else is something

[12] I realize as fully as my Canadian friends the unfortunate element in the common usage of "American" with reference solely to the United States rather than to North America. Canadians are of course as much Americans as we are. But, in point of fact, because United States is not a term easily put into adjective form, while America is, common usage has seized upon the last word of the official designation of this country rather than the first two, to use as the descriptive adjective for her citizens.

other than American racially. I can not see logically but that a person born in the United States in 1920 has just as much right to call himself an American as one born here in 1620. And further this right is just as logically passed on hereditarily now as it was then. In other words is there any reason not to accept little Gaetano Tomasino (let us say) as a friend and brother citizen, though his ancestors first landed on these fair shores in the early spring of 1920 instead of the late autumn of 1620, *provided*, and just as long as, *Gaetano behaves like an American?* If and when he does not then plainly something must be done about it. Practically, and in fact, that is the way in which Americans of all varieties, including that odd and rather dangerous creature now so much to the fore, the "100 per cent American," have come into being.

Divorce

Divorce is unquestionably a factor in the biology of human reproduction, and should therefore be considered in any general résumé of the subject. From the point of view of the present discussion, however, it is wholly impossible to do anything with this topic, since no data are available. In *Marriage and Divorce*, 1916, a special report of the Bureau of the Census issued in 1919, the following statement is made:

> Two investigations with respect to marriage and divorce have been made previously by the Federal Government—one in 1887–88 by the Department of Labor, covering twenty years from 1867 to 1886, inclusive, and the other, in 1906–07 by the Bureau of the Census, covering the twenty years from 1887 to 1906, inclusive. These two investigations together covered a consecutive period of forty years.

The results of the first of these studies are incorporated in the second. It thus results that the entire statistical knowledge of marriage and divorce for this country as a whole is contained in these volumes. And in no one of these are there any data about race or nationality. Consequently from our present viewpoint nothing can be done with the material.

IV. NATALITY

The most important data regarding births, from the viewpoint of the present discussion, have already been presented in the section on marriage, as a result of dealing with *effective* marriages solely. It remains here to consider certain special topics not there dealt with. These topics are (*a*) fertility, (*b*) illegitimacy, (*c*) stillbirths.

A. Fertility

The general fact that foreign-born women are much more fertile, under American conditions, than native-born is well known, and can be shown in many different ways. Perhaps the most striking way to set forth the broad fact is to show the relation of births to married women of child-

TABLE 52

Showing the relation between the number of foreign-born women of child-bearing age and their contributions to the population

STATE	PER CENT OF			
	Children of foreign-born mothers to total children of white mothers			Foreign-born married females 15 to 44 to white married females 15 to 44
	1919	1918	1917	1910
Rhode Island	*	53.59	55.76	49.94
Massachusetts	52.58	53.20	55.36	48.87
Connecticut	56.76	58.20	60.21	46.36
New York	48.99	49.10	51.64	42.71
Minnesota	21.90	22.25	24.86	33.99
New Hampshire	37.78	38.51	40.81	32.69
Pennsylvania	33.88	34.52	36.33	27.77
Michigan	29.11	30.08	32.46	26.45
Wisconsin	18.70	19.08	20.62	26.40
California	28.04	†	†	23.74
Washington	19.86	20.16	21.69	23.24
Maine	25.13	24.24	25.92	21.89
Utah	12.73	13.17	14.56	18.83
Vermont	20.71	21.38	22.47	18.11
Ohio	21.71	21.87	23.11	15.51
Oregon	12.98	†	†	14.81
Maryland	13.15	13.16	14.29	13.11
Kansas	6.54	6.12	6.91	8.12
Indiana	8.43	8.46	8.95	5.72
Virginia	2.06	2.06	2.05	2.35
Kentucky	0.82	0.77	0.86	1.55
South Carolina	0.58	†	†	0.90
North Carolina	0.30	0.23	0.34	0.47

* Excluded because all transcripts of birth certificates for the year were not received.
† Not added to registration area until later date.

bearing age in the population as a whole. This is done in table 52, which is in rearranged form, a table given on page 10 of *Birth Statistics*, 1919.

From table 52 it is seen that in twelve of the twenty-three states in the table the foreign-born women were the mothers of a higher percentage of

children than would be expected if they were no more fertile than the native-born women of the same age groups. In the case of Connecticut the percentage of births rises to 10 or more points higher than the percentage of representation in the population.

For one state, New York, Eastman[13] has made a very careful and thorough study of the birth-rates by race. Table 53 shows some of his chief results.

Commenting on this table Eastman says:

The features of interest that at once attract the attention are the extraordinarily high rates of the Italians, Russians and Austro-Hungarians (which amount to from 100 per cent to almost 200 per cent higher here than in the mother countries), and the exceptionally low rates of the Irish and Germans. A study of the statistics of immigration as contained in the reports of the Commissioner General of Immigration of the United States will reveal the probable cause of these remarkable differences. Of the total number of Italians, Austro-Hungarians and Russians, who were admitted into this country since July 1, 1880, 75.3 and 77 per cent respectively, arrived here from July 1, 1900, to June 30, 1914, while of the total number of Germans admitted since 1880, 60 per cent arrived prior to June 30, 1890, and only 19.4 per cent since July 1, 1900. The exact data regarding the Irish were not obtainable from the records from which the above figures were extracted, but it is very probable that the percentages are more or less similar to those of the Germans, assuming that the difference of age upon arrival does not differ materially between the two nationalities. This means that the three nationalities in New York State first mentioned are mostly composed of young adults in the most productive period of life, while the Germans and Irish are mostly people past childbearing age, or at least in the later stages of that period when the birth rate is much lower. It is notable that the Canadians and British have a lower birth rate in New York than in their respective countries of birth. This may be due to the greater similarity of their age constitution to that of the native class and to their greater inclination and ability to adopt the American standard of living.

Attention is directed to the fact that 73.1 per cent of all births to foreign-born women were to Italian, Russian and Austro-Hungarian mothers and that these races accounted for nearly 27 per cent of all the births occurring in the State outside of New York City, although they furnished less than 7 per cent of the total population.

From a strictly scientific standpoint these figures are not thoroughly reliable. For all practical purposes, however, there seems to be no adequate reason why they should not be used. If anything the birth rates of the foreign-born are probably too low, since it is likely that the percentage of each foreign nationality to the total population was not as great in 1916 as it was in 1910, owing to the practical cessation of all immigration during the years of 1915 and 1916. This would probably more than offset any increase that may have occurred in the foreign population of the State from an influx of these people from other states, attracted by better industrial conditions, etc. It may therefore be assumed, that whatever error there may be, would, if corrected, but emphasize the point that the birth-rate of native women in New York State is undoubtedly as low, or lower than that of France, and that the birth rate of the foreign-born woman is almost twice as great as that of the native woman.

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[13] Eastman, P. R., A comparison of the birth rates of native and of foreign-born white women in the State of New York during 1916, *Bulletin New York State Dept. of Health*, 15 pp.

TABLE 53

Births to white mothers, according to nativity of mothers, in New York State, (exclusive of New York City) in 1916 (after Eastman)

NATIONALITY OF MOTHER	ESTIMATED POPULATION IN 1916	NUMBER OF BIRTHS	NUMBER OF BIRTHS PER 1,000 ESTIMATED POPULATION	CRUDE BIRTH RATE OF NATIVE COUNTRY ACCORDING TO LAST REPORT BEFORE WAR	DATE OF LAST REPORT BEFORE THE WAR
Total white	4,643,786	102,834*	22.1		
Native white	3,777,685	64,889	17.2		
Foreign-born white	866,101	37,914	43.8		
English, Scotch, and Welsh	97,695	1,869	19.1	24.1† 25.5‡	1913
Irish	124,467	1,879	15.1	22.8	1913
German (includes German Poles)	171,435	2,421	14.1	27.5	1913
Italian	141,845	12,998	91.6	31.7	1913
Russian (includes Finland and Russian Poland)	82,195	7,281	88.6	44.0	1909
Austro-Hungarian (includes Austrian Poles)	81,256	7,307	89.9	31.3§ 36.3¶	1912
Canadian	104,270	2,219	21.3	24.0‖	1913
Other foreign-born	62,938	1,940	30.8		

* Nativity of mother unknown in the case of 31 births.
† England and Wales.
‡ Scotland.
§ Austria.
¶ Hungary.
‖ Province of Ontario.

TABLE 54

A comparison of the crude, legitimate and illegitimate birth rates of native and foreign-born mothers (from Eastman)

	BIRTH RATES		
	Births to native-born mothers	Births to foreign-born mothers	Excess percentage of births to foreign-born mothers
Number of births per 1,000 inhabitants	17.2	43.7	154
Number of legitimate births per 1,000 married women 15 to 44 years of age	137.1	253.2	85
Number of illegitimate births per 1,000 single, widowed or divorced women 15 to 44 years old	2.1	3.2	52

The above birth rates (17.2 for native women and 43.8 for foreign women) are not fairly comparable owing to the great differences between the age constitution of each element. There is not only a greater proportion of married women from fifteen to forty-five years of age among the foreign class (according to the 1910 U. S. Census it amounted to 70 per cent as against 52 per cent for native women), but a larger percentage of them are between the ages of twenty-one and thirty, the period of greatest productivity.

To illustrate the point Eastman presents the table, here given as table 54.

To indicate the reliability of the New York figures Eastman compares them with Kuczynski's data for Massachusetts in 1895. The results are shown in table 55.

It is clear that the agreement is close. The same result has also been got by Hill.[14]

TABLE 55

A comparison of the birth rates in Massachusetts in 1895 and in New York in 1916 (from Eastman)

	MASSACHUSETTS 1895	NEW YORK 1916
Births per 1,000 native population	17.03	17.2
Births per 1,000 foreign-born population	52.16	43.7
Births per 1,000 native adult female population	48.78*	48.6†
Births per 1,000 foreign-born females	107.29*	104.2†
Births per 1,000 married native women of child-bearing age	142.47*	137.1†
Births per 1,000 married foreign-born women	251.76*	253.2†

* Ages 14 to 50.

† Ages 15 to 44.

Altogether there can be no doubt that in those parts of the country where a relatively large proportion of the population is foreign-born, the fertility of the foreign-born women is greatly in excess of that of the native-born. Almost, if not quite, the first biological result of Americanization is to reduce the fertility of the marriages. This is, of course, to be expected on all of the following grounds:

a. The young immigrant woman is, generally speaking, a strong, vigorous, healthy animal—pioneering stock in fact. Even in these times it requires a person of more than the average gumption—both of body and of character—to pull up roots in the homeland and venture to a new world.

b. The standard of living of the immigrant is relatively low, and this has virtually always and everywhere meant a high birth rate.

[14] Hill, J. A., Comparative fecundity of women of native and foreign parentage in the United States, *Quart. Publ. Amer. Stat. Assoc.*, vol. 13, pp. 583–604, 1913 (N. S., no. 104).

c. The first broad economic result of Americanization is a raising of the standard of living. This makes itself felt in the first generation (native-born of foreign parents) in respect of the birth rate.

It would appear that this particular phase of the general problem of immigration, which causes some persons such great alarm, is one which in a generation or two adjusts itself quite automatically. Foreign-born women in this country do have a high birth rate, but their descendants very quickly slough off this attribute of barbarity, as it is regarded by some.

B. Illegitimacy

While having no direct bearing upon our main problem, the question of illegitimate birth rates is an interesting one on its own account. It has been by many persons considered to be an index of morality, but most statisticians who have studied the question are inclined to question or deny entirely its significance in this regard. Eastman has shown (*cf.* table 54 *supra*) that the illegitimate rate, *on the basis of single, widowed and divorced women*, is higher for the foreign than the native-born women in New York State. But it is also of interest to reckon the illegitimate rate on the basis of 1000 *total births*. The result of this method for the B. R. A. in 1919 is shown in table 56, which is modified from a table in *Birth Statistics*, 1919, pages 16 and 17. Those states from which the returns are known to be incomplete are omitted, but their results are included in the figures for the Registration Area as a whole.

The illegitimate rate per 1000 total births is more than three times higher for native-born than for foreign-born mothers as a whole. For no one of the groups of foreign-born mothers does the illegitimate rate on this base approach in magnitude that for the native-born mothers. In the separate states the few exceptionally high rates for foreign-born mothers are based in every case upon fewer than 5 illegitimate births, in which case, of course, no reliance is to be placed upon the single rate by itself.

These facts disclose an interesting statistical paradox. Table 54 shows that the illegitimate rate is higher per 1000 foreign-born women capable of having an illegitimate baby, than it is per 1000 native-born women in a similar social situation. But per 1000 total births the illegitimate rate is smaller for foreign- than for native-born mothers. The explanation is, of course, simple. There are so many more total births per 1000 foreign-born child-bearing women than per 1000 native-born that the illegitimate rate per 1000 births is bound to be low because the denominator of the rate fraction is large.

TABLE 56

Number of illegitimate births (exclusive of stillbirths) per 1000 total births for white children, by country of birth of mother, 1919

(Numbers are shown *in italics* where illegitimate births are less than 5)

AREA	COUNTRY OF BIRTH OF MOTHER													
	Total	United States	Foreign country											
			Total	Austria; includes Austrian Poland	Hungary	Canada	Denmark, Norway and Sweden	England, Scotland and Wales	Ireland	Germany; includes German Poland	Italy	Poland (not specified)	Russia; includes Russian Poland	Other foreign countries
Registration Area	12.0	14.9	4.1	3.9	5.1	10.0	5.5	8.6	8.7	5.6	1.5	4.1	2.1	3.7
Connecticut	9.1	15.0	4.4	6.3	*3.7*	8.9	11.2	*6.7*	4.7		2.5	7.5	3.1	*4.0*
District of Columbia	18.3	12.4	*2.6*								*11.8*			
Indiana	11.6	12.3	2.3	3.7	*5.4*			*4.9*		*2.7*		*1.2*		
Kansas	5.8	5.9	2.2					*9.3*					*2.3*	*3.8*
Kentucky	9.9	9.9	*2.3*			*47.6*								
Maryland	18.6	20.9	3.0	*2.4*			*25.0*			*12.3*		*2.5*	*1.5*	*14.7*
Michigan	12.7	15.5	5.5	5.6	*2.4*	9.0	10.3	15.6	*11.0*	5.8	*1.6*	3.7	2.3	5.5
Minnesota	16.0	18.8	5.7	3.4	*3.7*	10.8	6.2	19.1		*3.3*		*7.5*	*4.5*	6.3
New York	9.3	14.7	3.8	3.8	6.2	20.1	4.9	12.0	9.1	5.6	1.2	5.6	1.8	6.0
North Carolina	14.8	14.1												
Ohio	13.1	15.7	3.5	3.4	4.9	12.7	*5.5*	5.7	*4.0*	7.5	*1.1*	*1.1*	2.0	4.1
Oregon	13.6	14.5	5.8			*11.1*		*6.0*		*7.2*	*13.2*		*14.2*	*3.1*
Pennsylvania	16.3	22.1	4.6	4.1	6.4	*11.1*	10.4	10.0	12.7	9.1	1.9	5.6	3.3	5.3
South Carolina	16.9	16.9												
Utah	7.8	8.4	3.7				*5.1*	*2.9*		*13.7*				*2.4*
Virginia	18.2	18.5												
Washington	9.5	10.6	5.3	*6.7*		14.9	4.9	*4.3*	*7.6*	*3.8*	*3.1*			*1.5*
Wisconsin	11.8	13.4	3.9	3.2	*5.3*	*4.6*	*3.1*	*5.4*		6.1	*1.3*	5.3	*1.6*	*3.7*

The essential point is that brought out in table 54. The foreign-born, unmarried, widowed or divorced woman is more likely to have a child than a similarly situated native-born woman. This probably does not mean better morals on the average in the latter case, but simply better judgment, which in turn means higher intelligence.

C. Stillbirths

Data for stillbirths are not particularly accurate in the United States because of local differences in the legal definition of a stillbirth. Regarding this matter the Census Bureau (*Birth Statistics*, 1918, pp. 28 and 29) makes the following comments:

> Interpretation of the data relating to stillbirths must be made with extreme caution, as the term "stillbirth" is not used in the same sense in the various states and the percentage of the completeness of these reports is not known. The Model Law reads: A stillborn child shall be registered as a birth and also as a death, ... provided that a certificate of birth and a certificate of death shall not be required for a child that has not advanced to the fifth month of uterogestation.
>
> Of the states in the birth registration area, Minnesota, New York, and North Carolina have similar provisions in their laws. The District of Columbia requires a certificate for a fetus "passed the fifth month." The Indiana law reads "seven months' gestation and over," and the remaining states use the term stillbirth, but do not define it.
>
> In Maryland special efforts have been made to secure uniform registration of stillbirths of even the earlier months of uterogestation, so that the data for this state are not comparable with those for other states. At first thought these differences in the various state laws seem to limit greatly the comparability of the data, but the fact that the figures of those states requiring reports only of children which have reached the fifth month approximate the figures of those states which do not define the term "stillbirth," warrants the conclusion that in the latter states only stillbirths of the later months are for the most part reported, so that the figures are fairly comparable. Moreover, it is undoubtedly true that stillbirths of the later months of uterogestation are well reported, because such stillbirths are interred, and burial cannot legally take place without a burial permit, issued upon receipt of the physician's certificate of death.
>
> All data relating to stillbirths have been compiled from transcripts of stillbirths reported as births. Undoubtedly the total numbers of stillbirths would have been somewhat larger had data been compiled from transcripts of stillbirths reported as deaths, for foundling stillbirths would then have been included; but as most of the important facts relating to stillbirths are recorded on the birth certificates only, the Bureau of the Census gathered the information from this source.

For reasons, presumably connected with economy (most certainly of the false variety), all data on stillbirths are omitted in the 1919 *Birth Statistics*. We shall therefore have to take 1918 for the latest figures.

Table 57 (from *Birth Statistics*, 1918, in rearranged form) gives the data as to stillbirths by country of birth of mother. To this table has been

added a final column giving the average total fecundity of the several groups of mothers, as of the same year. The Registration Area is not quite the same for the figures of the last column and the next to the last, but the error so introduced is negligible for the use to which the figures are here put.

The stillbirth rate is higher, on the basis of total live births, for foreign- than for American-born women in general. In the case of Scandinavian, German, Polish and Hungarian mothers the stillbirth rate is lower than

TABLE 57

Births, stillbirths, stillbirths per 100 live births, and average number of children ever born, for white children by country of birth of mother, 1918

COUNTRY OF BIRTH OF MOTHER	BIRTHS	STILL-BIRTHS	STILL-BIRTHS PER 100 LIVE BIRTHS	AVERAGE NUMBER OF CHILDREN EVER BORN
United States	838,000	30,891	3.7	3.1
Total foreign	314,780	12,392	3.9	3.9
Canada	13,861	651	4.7	3.5
Ireland	14,038	620	4.4	3.3
England, Scotland and Wales	11,069	473	4.3	3.3
Italy	71,373	2,989	4.2	4.4
Russia (includes Russian Poland)	60,269	2,369	3.9	3.3
Austria (includes Austrian Poland)	62,111	2,384	3.8	3.9
Other foreign countries	22,355	835	3.7	3.6
Denmark, Norway and Sweden	11,624	413	3.6	3.6
Germany (includes German Poland)	15,414	559	3.6	4.6
Poland (not specified)	14,534	525	3.6	4.2
Hungary	18,132	574	3.2	3.8

for the native-born mother. One's first thought is that the higher stillbirth rate of the other foreign-born mothers, as compared with the native-born, is due to the higher fertility of the former group. Comparing the fourth with the third column of table 57 gives little support, however, to such a view.

On the contrary, figure 32, which is plotted from the data of table 57, seems rather definitely to indicate that the opposite relation holds on the whole. Namely, the more children a woman has had the less likely is the next birth to be a stillbirth.

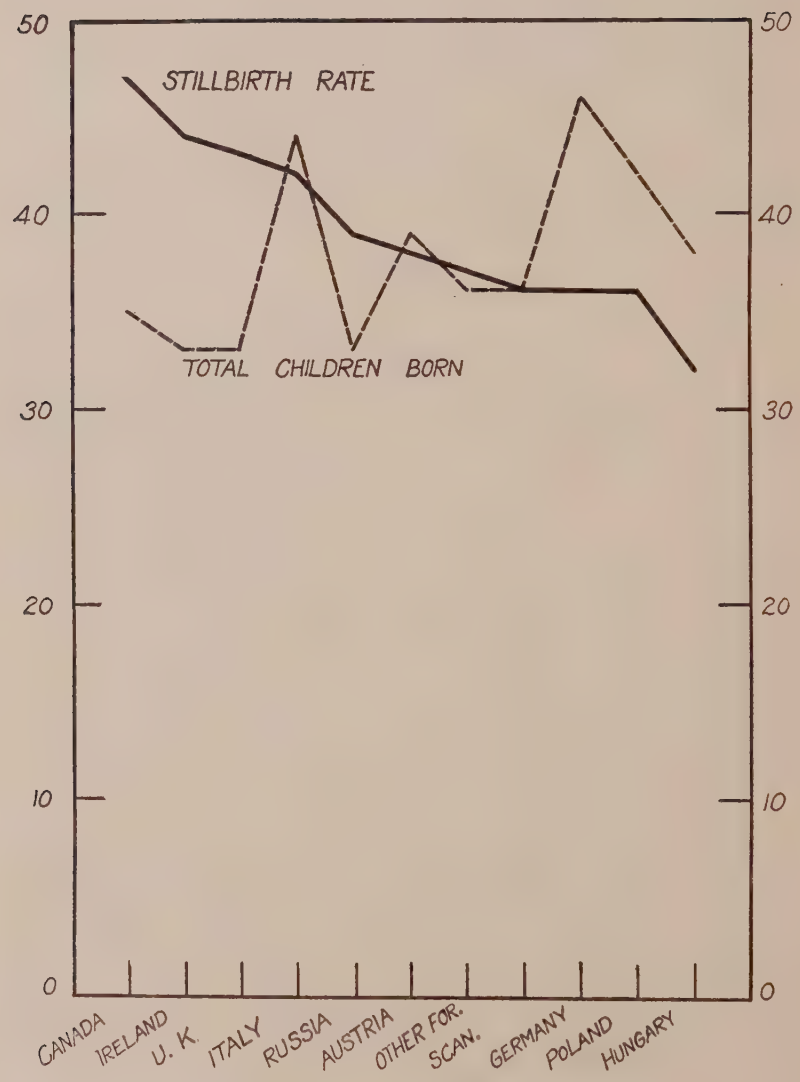

FIG. 32. SHOWING THE RELATION BETWEEN THE STILLBIRTH RATE AND THE TOTAL NUMBER OF CHILDREN WHICH HAVE BEEN BORN

V. MORTALITY

From published official data it is impossible to get any comprehensive or precise information as to the relative mortality of the different racial groups in this country. The fault is not so much with the original data contained in the death certificates, but with the fact that this material is not compiled, and by publication made available for the public information. The most that can be done is to learn something about the comparative mortality of the native- and foreign-born as total groups.

For the purpose of indicating what the facts in this restricted field show, tables 58 and 59 have been prepared. Table 58 shows, for the six states, Connecticut, Massachusetts, Minnesota, New York, Pennsylvania and Wisconsin taken together, the population and deaths in 1910, divided according to whether the decedents were (*a*) native-born of native parents (*b*) native-born but of foreign or mixed foreign and American parentage, and (*c*) foreign-born, and also by sex and age.

The six states chosen were taken because of their representative character in respect of different industries, and because of their large foreign-born populations. Together the states included in table 58 had in 1910 a total population of 25,205,393 individuals about each of whom age, sex, nativity, and parent nativity were definitely known. This is a respectable total number, and tends to give confidence in the results.

From the data of table 58 have been calculated death rates specific for age and sex for each of the three groups. These death rates are exhibited in table 59. Unless some arithmetical error has been committed in these calculations and overlooked, they represent a very close approximation to the true forces of mortality, operating in each defined group. The reader should not confuse them with crude death rates. They are true specific death rates, the specificity extending to the three variables age, sex and nativity with parent nativity.

The data of table 59 are shown graphically in figures 33 and 34.

The first point which strikes one from the examination of these specific death rates is that the infant mortality (death rate under 1) is generally *lower* for the foreign-born than for either of the other two classes. This result is a spurious one, arising from the following circumstances. To get entered in the deaths under 1, in this country, a foreign-born baby must have first been born abroad, survived the trip to this country and then died here. Now in view of the fact that the heaviest incidence of mortality in the first year of life is known to be always and everywhere in the first month of the first year, it necessarily follows that some un-

TABLE 58

Population and deaths by sex, age and nativity, in six selected states, 1910

AGE	NATIVE-BORN MALES OF NATIVE PARENTAGE		NATIVE-BORN FEMALES OF NATIVE PARENTAGE		NATIVE-BORN MALES OF FOREIGN OR MIXED PARENTAGE		NATIVE-BORN FEMALES OF FOREIGN OR MIXED PARENTAGE		FOREIGN-BORN MALES		FOREIGN-BORN FEMALES	
	Population	Deaths	Population	Deaths	Population	Deaths	Population	Deaths	Population	Deaths	Population	Deaths
Under 1 year	133,312	17,414	129,877	13,284	151,852	24,298	147,861	18,403	1,678	202	1,546	190
Under 5	640,590	23,449	626,017	18,651	684,231	34,440	671,771	28,547	24,433	705	23,852	654
5–9	572,115	1,996	560,499	1,802	563,975	2,229	558,630	2,103	77,482	303	76,590	288
10–14	536,371	1,210	528,686	1,165	526,755	1,220	521,939	1,130	97,286	241	96,169	232
15–19	509,528	1,637	516,632	1,559	486,348	1,798	496,577	1,553	188,413	901	189,223	636
20–24	464,304	2,062	487,573	1,977	378,932	2,169	408,925	1,966	421,031	2,470	352,597	1,508
25–29	412,284	1,985	420,361	2,000	300,913	2,182	323,635	2,011	496,717	2,963	370,220	1,985
30–34	353,853	1,815	355,736	1,687	263,865	2,642	284,009	2,032	438,641	3,049	326,409	2,086
35–39	324,381	1,981	319,080	1,619	252,168	2,945	267,062	2,127	400,235	3,647	312,995	2,439
40–44	270,568	1,872	266,494	1,653	201,219	2,713	212,287	2,020	359,062	4,170	279,084	2,566
45–49	233,522	1,951	230,752	1,689	166,141	2,669	173,051	1,966	303,204	4,618	242,098	2,872
50–54	217,332	2,558	214,152	2,213	135,964	2,652	137,891	2,001	234,597	4,651	192,357	3,180
55–59	172,234	3,018	170,721	2,512	75,189	2,027	76,818	1,593	163,464	4,687	147,355	3,577
60–64	141,568	3,664	149,246	3,007	39,264	1,411	42,071	1,209	139,908	5,631	136,559	4,918
65–74	186,961	8,438	207,898	8,240	33,910	1,913	38,058	1,952	175,063	11,388	178,827	11,079
75–84	68,992	6,785	85,992	7,749	7,346	905	9,144	1,001	61,395	7,944	67,783	8,478
85–94	10,448	1,955	15,356	2,765	1,045	271	1,431	397	8,883	2,298	10,845	2,586
95 and over	285	92	591	181	35	13	48	13	349	156	498	208
Totals....	5,115,336	66,468	5,155,786	60,469	4,117,300	64,199	4,223,347	53,621	3,590,163	59,822	3,003,461	49,292

known, but fairly large proportion of the new-born babies of families about to immigrate to the United States in 1910 died, either before the family left the old country, or while they were upon the ocean. Consequently they would never appear in our statistics. But there would be left to die in this country during the first year of life a smaller number of foreign-born babies, and furthermore a selected kind of babies, namely, the survivors of the period of heaviest incidence of infant mortality. So we must conclude that the foreign-born mortality under 1 is not

TABLE 59

Specific death rates per 1,000 living of same sex and age, from Table 58

AGE	NATIVE WHITE OF NATIVE PARENTAGE		NATIVE WHITE OF FOREIGN OR MIXED PARENTAGE		FOREIGN BORN WHITE	
	Male	Female	Male	Female	Male	Female
Under 1 year	130.63	102.28	160.01	124.46	120.38	122.90
Under 5	36.61	29.79	50.33	42.53	28.85	27.42
5–9	3.49	3.21	3.95	3.77	3.91	3.76
10–14	2.26	2.20	2.32	2.17	2.48	2.41
15–19	3.21	3.02	3.70	3.13	4.78	3.36
20–24	4.44	4.05	5.72	4.81	5.87	4.28
25–29	4.81	4.76	7.25	6.21	5.97	5.36
30–34	5.13	4.74	10.01	7.15	6.95	6.39
35–39	6.11	5.07	11.68	7.96	9.11	7.79
40–44	6.92	6.20	13.48	9.51	11.61	9.19
45–49	8.35	7.32	16.07	11.36	15.23	11.86
50–54	11.77	10.33	19.51	14.51	19.83	16.53
55–59	17.52	14.71	26.96	20.74	28.67	24.27
60–64	25.88	20.15	35.94	28.74	40.25	36.01
65–74	45.13	39.63	56.41	51.29	65.05	61.95
75–84	98.35	90.11	123.20	109.47	129.39	125.08
85–94	187.12	180.06	259.33	277.43	258.70	238.45
95 and over	322.81	306.26	371.43	270.83	446.99	417.67

accurately comparable with the native-born mortality in the same age group. The influence of this spurious element makes itself felt throughout the "Under 5" group.

After this period is passed the specific rates are *lower*, at all ages right away to the end of life, for the native-born of native parents than for either of the other two groups, in both males and females, with the single exception that the native-born of foreign or mixed parentage rate for females in the age group 95 and over is a little lower than the rate for native-born women of native parentage. No significance is to be attached to this

slight deviation from the rule at the end of the curve, because all rates for that age group are subject to large probable errors because of the meager numbers involved.

The superior estate of the native-born of native parents in respect of mortality undoubtedly arises from the combined action of the following factors:

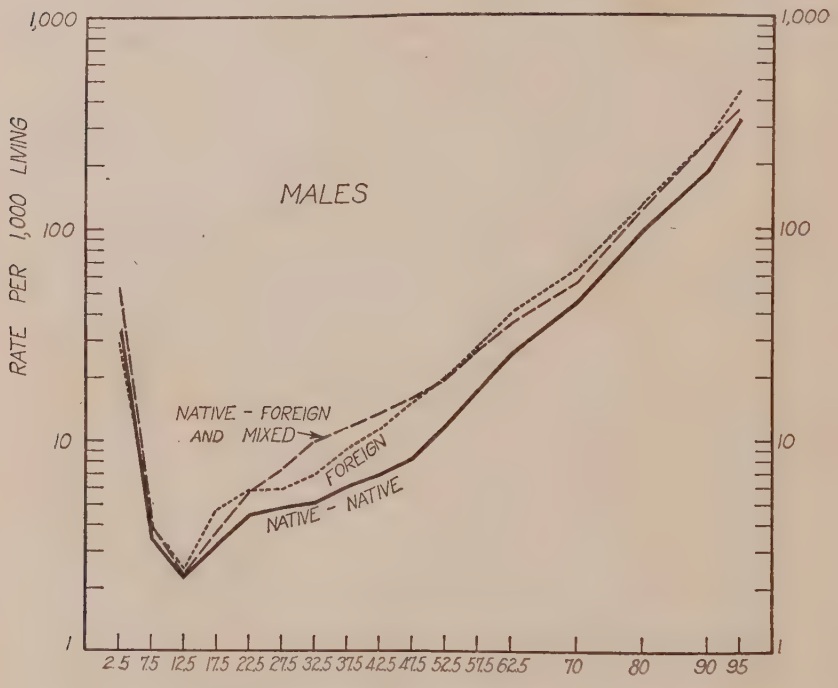

FIG. 33. SPECIFIC DEATH RATES FOR (*a*) NATIVE-BORN MALES OF NATIVE PARENTAGE (SOLID LINE), (*b*) NATIVE-BORN MALES OF FOREIGN OR MIXED PARENTAGE (DASH LINE), AND (*c*) FOREIGN-BORN MALES (DOT LINE) IN SIX SELECTED STATES (SEE TEXT) IN 1910

a. Superior intelligence, not perhaps in an absolute or abstract sense, but concretely in respect of awareness of and ability to grapple with the particular kind of environmental difficulties connected with living in the United States. This is probably the most important single factor. The native-born American of native parentage has a pretty fair fund of knowledge and tradition as to how best to contend with strictly American dangers—hygienic, economic and other.

b. Superior physical environment, associated with a higher general standard of living. If one were obliged to put into a single statement the most general and important reason why genuine immigrants (meaning those intending to stay) now come to our shores, it would run, I think, in about this form: That above everything else the immigrant desires and intends to raise the standard of living of his family and descendants.

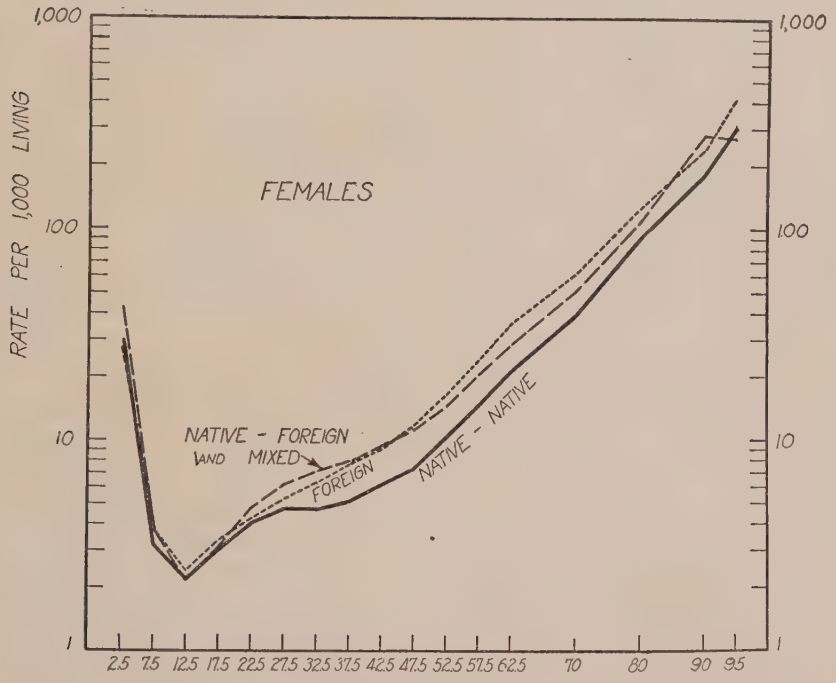

FIG. 34. SPECIFIC DEATH RATES FOR (*a*) NATIVE-BORN FEMALES OF NATIVE PARENTAGE (*b*) NATIVE-BORN FEMALES OF FOREIGN OR MIXED PARENTAGE, AND (*c*) FOREIGN-BORN FEMALES IN SIX SELECTED STATES IN 1910

Significance of lines as in figure 33

After two generations have been born in this country the result has either been achieved or it has not. If it has not, the family that started the enterprise has, generally speaking, either died out or gone back home. There can be no question about the fact that, by and large, and in most parts of the country, the standard of living of the native-born of native parentage is definitely higher than that of either of the other two groups.

On the other hand, the relations of the specific mortality curves are, for the native-born of foreign or mixed parentage, most curious, and not entirely easy to explain satisfactorily. Taking the male data first, we see that in youth (from age 10 to about age 20) the specific rates are higher for the foreign-born. The curves cross at about age 22.5, and all through young adult and early middle life—up to age 52.5—the specific rates for the foreign-born are lower than those of the native-born of foreign or mixed parentage. From then on to the end of the life span the specific rates for the foreign-born are again higher than those for the native-born of foreign or mixed parentage.

The pertinent factors in the case appear to be as follows:

1. During childhood the foreign-born individuals probably receive on the whole less effectively good care in a hygienic sense than native-born children of parents of foreign or mixed parentage, because recent immigrants understand our environment less well. Furthermore there is an extra occupational hazard upon foreign-born lives even in the childhood ages. So then the higher specific rates of childhood on the foreign-born are exactly what we should expect.

2. During early adult and middle life we have, apparently:

	FOREIGN-BORN	NATIVE-BORN OF FOREIGN OR MIXED PARENTS
a. Physical environment and standard of living	Worse	Better
b. Occupational hazard	Worse	Better
c. Innate biological constitution	Better	Worse
d. Racial constitution of the population	Better	Worse

Rubrics *a* and *b* need no explanation or argument, so far as I can see. Rubric *c* is meant to express the fact that immigration, even here and now, is a real form of pioneering. Only the relatively superior—in comparison with the group *out of which they come*—in physique, courage and general mental soundness have the initiative and strength of character to undertake the big adventure of starting anew in a strange country. That this selective action has its effect upon mortality, and that this effect is a considerable one, admits of no doubt.[15]

[15] *Cf.* Chapters X and XVII of this book; also Pearl, R., *The Biology of Death*, Philadelphia (J. B. Lippincott Co.), 1922, especially the chapter on inheritance of duration of life.

Under rubric *d* is included whatever differences in race stock there may be between the foreign-born and the native-born of foreign or mixed parentage. Dublin and Baker[16] consider, on the basis of a study of New York and Pennsylvania alone, that the difference in the death rates that we are now discussing is practically entirely due to the "predominance of the Irish, German and British stocks among the first generations of Americans" in the age period 25 to 44. That this is a factor admits of no doubt. And it is an innate, biological one in last analysis. It does not seem to me, however, that Dublin and Baker have entirely demonstrated that it is the only, or even overwhelmingly important factor in the case.

In the study of the mortality of different race stocks in this country lies a wonderful opportunity for a piece of analytical work, the results of which could not fail, if it were carefully and sagaciously done, to throw a flood of light on some of our most pressing social as well as public health problems. What is wanted here is quantitative fact, not opinion. And it can be got, if only somebody sufficiently interested and public-spirited will finance the business.

So far we have been focussing attention upon the male curves. If we now turn to the female data, however, it is immediately apparent that whatever differences appear are essentially of degree not kind. In their general course the three female curves are essentially like the three male curves.

Altogether it may fairly be said that the exhibition of specific death rates, meager as is the specificity in respect of race, has served to define a large and important problem. If we could get compilations of deaths and populations separately for each racial group by age, sex, occupation, nativity and parent nativity, how much farther we could carry our knowledge of the factors influencing human mortality.

Already the attempt has been made to carry the analysis of racial factors in mortality in this country farther. Dublin and Baker (*loc. cit.*), by means of data specially obtained from the Census Bureau, have recently studied in a most interesting and profitable manner the mortality of different race stocks in New York and Pennsylvania. Dublin[17] earlier had made a similar study for certain race stocks in New York.

[16] Dublin, L. I., and Baker, G. W., The mortality of race stocks in Pennsylvania and New York, *Quart. Publ. Amer. Stat. Assoc.*, March, 1920.

[17] Dublin, L. I., Factors in American mortality, *Amer. Econ. Rev.*, vol. 6, no. 3, September, 1916.

Dublin and Baker examined separately certain important causes of death. Their findings were as follows:

Tuberculosis of lungs. The outstanding feature relative to pulmonary tuberculosis is the very unfavorable position of the Irish. Both sexes suffer extraordinarily from this disease during the greater part of life (25 to 84). At 25 to 44 the rates for Irish males are twice as high as for natives, and in the age period 45 to 64 the excess is even greater. The death rate among Irish males from tuberculosis continues high even at the advanced ages. The facts with regard to the Irish in the two states studied, are very different indeed from those in the native country, both as to the high rates obtained and the general configuration of the mortality curve. German and British males also show higher tuberculosis rates than the native-born at the older age. On the other hand, the Italians, Russians and Austro-Hungarians present comparatively low tuberculosis death rates. At most of the age periods they are even lower than for the native-born. Italian females in New York show a striking exception to the usual rule in that their rates are higher than those for males, especially in early life. Otherwise there are no important differences in the several race groups with reference to this disease.

Cancer. Practically every foreign race group shows higher cancer rates than do the native-born. Italian males and females offer the only clear-cut exceptions At the age period 45 to 64, foreign-born males show rates from 50 to 100 per cent higher. The conditions among the Irish are strikingly bad. Among females at ages 45 to 64, the Germans and the British also show higher rates than do the native-born, but the excess is not as marked as it is among the Irish.

Organic diseases of the heart. Again the Irish present the highest rates, followed by the British and the Germans. The conditions prevailing among those from Austria-Hungary, Russia and Italy are, on the whole, very favorable with regard to this disease. It is very difficult to understand the reason for the aberrant death rates among the Irish in Pennsylvania and New York, for they are so much higher than are those for the native stock and for the Irish in their own country.

Pneumonia, all forms. From ages 45 to 84, the rates of the native-born are much the lowest. The Irish are again under the greatest handicap, their rates being about three times as high at ages 45 to 64. The rates for Italians are very unfavorable also at ages 45 to 84. The disadvantage of the Germans, British and Russians is less among females than among males. The position of the Austro-Hungarians is very much the same as the native-born at 25 to 64.

Bright's disease. Again we must record the very unfavorable position which the Irish occupy. From 25 to 84 years of age, the rates for this nationality are about 100 per cent higher than for the native-born; among females, the excess is even greater. At ages 45 to 84, the rates from Bright's disease are high for Austro-Hungarians, Russians, Germans and British. The position of the Italians is quite favorable.

Violence (excluding suicide). Deaths from this cause are largely from accident, and we find, of course, that the positions of the several race groups are determined by the extent to which they engage in occupations that expose them to such dangers. The figures are especially striking for Pennsylvania where large numbers of immigrants find employment in mills and mines. Males of all the important foreign groups show very high rates from this cause, the worst conditions prevailing among the Austro-Hungarians, followed by the Irish and Italians.

Their whole study Dublin and Baker summarize as follows:

1. Of the three main groups of the white population in Pennsylvania and in New York, (*a*) native-born, of native parents, (*b*) native-born of foreign or mixed parentage and (*c*) foreign-born, the first has the lowest mortality. This is true for both sexes and for virtually every age period, but it is most marked at the adult ages.

2. The foreign-born and the native-born of foreign or mixed parentage, agree much more closely with each other than with the native stock. An interesting exception presents itself, however, at ages 25 to 44, at which period the foreign-born have a great advantage over the native-born of foreign or mixed parentage. The reason for this is the predominance of the Irish, German and British stocks among the first generation Americans at this age period. After age 45, these two groups of the foreign stock are of the same racial extraction, and their death rates are in very close agreement.

3. The death rates of the component groups among the foreign-born vary considerably. The Austro-Hungarians, Russians, and Italians present altogether favorable conditions, while the British, Germans and Irish show death rates very greatly in excess. This is especially true of the Irish, whose mortality is about double that of the native stock. The rates for the Germans, British and Irish are much higher in America than in their own countries. Pulmonary tuberculosis, pneumonia and degenerative diseases including heart disease, Bright's disease, and cancer are largely responsible for this unfavorable mortality.

4. The findings of the previous study for New York State are confirmed. The unfavorable conditions of life and work among foreign races to which attention was directed in the study for New York are found to prevail in Pennsylvania as well. The facts emphasize the necessity for special public health work for the people of foreign origin. The much more favorable economic conditions under which they live in the United States than in their own countries should result in lower death rates. But in several instances, we find that this does not prevail; the facts indicate on the whole, deterioration rather than improvement. Is it possible that our immigrants are not representative of the best in their native countries? It has often been supposed that the immigrants comprised the most vigorous among their own people; the results, however, do not confirm this impression, but suggest many questions for further inquiry.

5. It is very important that a study similar to this one be carried out as soon as the results of the 1920 census are available, to determine whether any differences of importance have appeared in the interval of ten years.

Interesting and valuable as is the study of Dublin and Baker it is plain that a great deal further remains to be done along similar lines.

A very interesting study has been made by Eastman[18] of the relation of parental nativity to the infant mortality of New York State. It brings out in such a clear manner the significance of what are perhaps the two most important elements in determining rates of mortality, namely, innate biological constitution, on one hand, and general intelligence, on the other hand, that it will be of value to examine the results in some detail. Tables 60 and 61 (which are tables V and VI of Eastman's paper) show the data.

[18] Eastman, P. R., The relation of parental nativity to the infant mortality of New York State, *Amer. Jour. Dis. Child.*, vol. 17, pp. 195–211, 1919.

TABLE 60

Infant mortality from principal causes and by the nativity of the mothers—1916 (from Eastman)

	TOTAL BIRTHS	DEATHS UNDER ONE YEAR FROM ALL CAUSES	INFANT MORTALITY	DEATHS FROM PRINCIPAL CAUSES									
				Communicable diseases (1–14 inc.)		Respiratory diseases (28-29 and 86–98 inc.)		Gastro-intestinal diseases (103-104 inc.)		Premature birth, congenital debility and malformations (150–153 inc.)		All other causes	
				Deaths	Infant mortality	Deaths	Infant mortality	Deaths	Infant mortality	Deaths	Infant mortality	Deaths	Infant mortality
Total mothers	103,530	9,912	95.7	471	4.5	1,530	14.8	2,265	21.9	4,435	42.8	1,211	11.7
Total white mothers	102,834	9,760	94.9	463	4.5	1,505	14.6	2,239	21.8	4,372	42.5	1,181	11.5
Total colored mothers	696	152	218.4	8	11.5	25	35.9	26	37.4	63	90.5	30	43.1
Native white mothers	64,889	5,648	87.0	229	3.5	684	10.5	1,096	16.9	2,943	45.3	696	10.7
Total foreign white mothers	37,914	4,112	108.4	234	6.1	821	21.6	1,143	30.1	1,429	37.7	485	12.8
Italian mothers	12,998	1,259	96.9	90	6.9	313	24.1	317	24.4	402	31.0	137	10.5
Russian mothers (excluding Russian Poland)	3,665	377	102.9	18	4.9	64	17.5	128	34.9	126	34.4	41	11.2
Austro-Hungarian mothers (excluding Austrian Poland)	6,345	822	129.6	41	6.4	165	26.0	273	43.0	268	42.3	75	11.8
Polish mothers (including German, Austrian and Russian Poland)	4,703	577	122.7	37	7.9	116	24.7	191	40.6	171	36.3	62	13.2
Total for group	27,711	3,035	109.5	186	6.7	658	23.7	909	32.8	967	35.0	315	11.4
British mothers	1,869	134	71.7	6	3.2	14	7.5	22	11.7	68	36.4	24	12.8
Irish mothers	1,879	182	96.9	8	4.2	19	10.1	40	21.3	82	43.7	33	17.6
German mothers	2,290	283	123.3	7	3.0	48	20.9	80	34.8	117	51.0	31	13.5
Canadian mothers	2,219	243	109.5	13	5.9	43	19.4	45	20.2	107	48.2	35	15.8
Other foreign-born mothers	1,940	235	121.1	14	7.2	39	20.1	47	24.2	88	45.4	47	24.2
Total for group	10,203	1,077	105.6	48	4.7	163	16.0	234	22.9	462	45.3	170	16.7

TABLE 61

Principal causes of deaths of infants under 1 year of age showing percentages due to certain elements of the population (from Eastman)

	TOTAL BIRTHS	BIRTHS PER CENT	TOTAL DEATHS UNDER ONE YEAR		CAUSES OF DEATH									
					Communicable diseases (1–14 inc.)		Respiratory diseases (28-29 and 86–98 inc.)		Gastro-intestinal diseases (103-104 inc.)		Prenatal causes, etc. (150–153 inc.)		All other causes	
			Deaths	Per cent	Deaths	Per cent	Deaths	Per cent	Deaths	Per cent	Deaths	Per cent	Deaths	Per cent
Total white mothers	102,834	100.0	9,760	100.0	463	100.0	1,505	100.0	2,239	100.0	4,372	100.0	1,181	100.0
Native white mothers	64,889	63.1	5,648	57.9	229	49.5	684	45.4	1,096	49.0	2,943	67.3	696	58.9
Total foreign-born mothers	37,914	36.9	4,112	42.1	234	50.5	821	54.6	1,143	51.0	1,429	32.7	485	41.1
Italian, Russian, Austrian and Polish mothers	27,711	27.0	3,035	31.1	186	40.2	658	43.7	909	40.6	967	22.1	315	26.7
Other foreign-born mothers	10,203	9.9	1,077	11.0	48	10.3	163	10.8	234	10.4	462	10.6	170	14.4

Commenting on table 60 Eastman says:

The striking dissimilarity existing in the relative importance of the several main groups of causes, as distinguished between the native- and foreign-born mothers, is at once apparent even on a casual review. The infant mortality from communicable diseases was almost 75 per cent greater among children of foreign mothers than among the babies of native mothers; from respiratory diseases it was over 100 per cent greater, and from gastro-intestinal diseases the excess was about 78 per cent; but the rate from prenatal and other causes peculiar to early childhood was higher among the native element by over 20 per cent. Pursuing the analysis still further, by dividing the total number of foreign-born mothers into the same two groups adopted in the preceding tables, we find these differences much accentuated when the native stock is compared with the group comprising the Italians, Russians, etc. The other foreign-born mothers, considered as a class, also exceeded the native mothers in the death rates from communicable, respiratory, and gastro-intestinal diseases, but the rates from prenatal, etc., causes were identical.

Although the table gives us a very fair idea as to the relative prevalence of the several causes among certain classes of the population, it fails to show the percentage of the complete number of deaths from any particular cause that was attributable to each of the three classes of the population.

This relation referred to is brought out in table 61. Regarding this Eastman says:

Considering first the deaths of children of native white mothers, it will be noted that although they formed 57.9 per cent of all the deaths from all causes, they nevertheless contributed only 49.5 per cent of the total deaths from communicable diseases, 45.4 per cent of all the deaths from respiratory diseases, and 49 per cent of the total mortality from gastro-intestinal diseases. At the same time, 67.3 per cent of all the deaths from premature birth, congenital debility, congenital malformations, accidents at birth, etc., were of children of these mothers. In contrast with these it is interesting to observe that, notwithstanding the fact that only 42.1 per cent of all the white infant deaths occurred to children of foreign-born mothers, 50.5, 54.6 and 51 per cent respectively, of the complete infant mortality from communicable, respiratory and gastro-intestinal diseases were referable to them. As to the mortality from prenatal causes, however, it is to be remarked that only 32.7 per cent of the total from these causes occurred among this element in the population. This table shows also that the high death rates from the first three groups of diseases, incident to the children of the foreign-born, were particularly attributable to the Italians, Russians, Poles and Austro-Hungarians.

We may therefore consider that our previous surmise has been corroborated, namely, that the chief causes of infant mortality among the native population originate for the most part in adverse prenatal conditions, but that among the foreign element, and especially the Italians, Poles, etc., the most frequent causes of death are communicable, respiratory and gastro-intestinal diseases.

The explanation of these results is undoubtedly in general terms as follows: The fewer deaths from prematurity and congenital defects among the children of Italian and Slavic mothers indicate that superiority

of innate biological constitution which is generally associated with emigrating stocks. On the other hand, the high death rate among these infants from causes which Eastman classes as communicable, respiratory and gastro-intestinal is chiefly due to the mothers' ignorance. As Eastman says:

> It is not to be wondered at that they are the dominant causes among the foreign-born population, the majority of whom are poor, illiterate, without knowledge of English and almost wholly ignorant of the elements of modern sanitation, and inhabit, as a rule, the most congested districts of the large manufacturing centers.

Guilfoy[19] has also made an interesting study of the influence of race upon mortality in New York City. His results are in good accord with those of the other investigators which have been cited.

VI. VITAL INDEX

The term "vital index" may be used to designate that measure of a population's condition which is given by the ratio of births to deaths within a given time. It may fairly be said that there is no other statistical constant which furnishes so adequate a picture as this of the net biological status of a population as a whole at any given moment. If the ratio 100 births/deaths is greater than 100 the population is in a growing and in so far healthy condition. If it is less than 100 the population is *biologically* unhealthy. Depopulation may not be actually occurring, if there is a sufficient amount of immigration to make up the deficiency in births. But fundamentally and innately the condition is not a sound one from a biological standpoint, though under certain circumstances it may be from a social standpoint. It is curious, in view of the obvious significance of this constant, the vital index of a population, that so little attention is paid to it by demographers. After much study of it (mostly unpublished) I am convinced that no single figure gives so sensitive a measure of the vitality of a nation, or any subgroup of people as this does. There appears to have been no adequate general discussion of it since that of Wernicke[20] in 1889, and even he does not use it in the most effective manner or form. Sundbärg[21] proposed the use of death rates as a "measure of civilization"

[19] Guilfoy, W. H., The death-rate of the city of New York as affected by the cosmopolitan character of its population, *Quart. Publ. Amer. Stat. Assoc.*, vol. 10, pp. 515–522, 1907 (N.S., no. 80).

[20] Wernicke, J., *Das Verhältniss zwischen Geborenen und Gestorbenen in historischer Entwicklung und für die Gegenwart in Stadt und Land*, Jena, 1889, vi, and 91 pp., 8 vo.

[21] Sundbärg, G., Dødstalen saasom Kulturmätare. *Nationalökonomiska Föreningens Förhandlingar*, i Aaret, 1895, Stockholm, 1896.

of different peoples. Rubin[22] criticized Sundbärg, but only in respect of technique, proposing as a measure of civilization D^2/B where D = deaths and B = births. Pell[23] has dealt with the idea implicit in the birth/death ratio, but in a most inadequate manner.

For the purposes and viewpoint of our present discussion this constant is peculiarly fitted. One of our main problems is to see what is to be the probable future course of the several racial elements in our population. No better indicator of this could be had than the vital indices of the different groups. Unfortunately we find ourselves restricted, just as in other parts of the discussion, by a lack of the raw data needful to undertake a properly detailed analysis. It will be necessary, as in all of the previous discussion, to make the best of a somewhat bad case.

In table 62 are shown four vital indices for urban, rural and total births and deaths of each state in the B. R. A. for the years 1915 to 1918 inclusive.

The significance of the several indices is as follows:

$$\text{Vital index } A = \frac{100\ (\text{Births of whites of native parents})}{\text{Deaths of all native whites}}$$

In this index the births and deaths come from a group of the population in which the births in the numerator and the deaths in the denominator come from the same kind of people in at least the one respect that they were born in the United States. Greater homogeneity than is indicated by this identity of birth place would be desirable but is impossible with existing official statistics. The children born were of course native, and their parents were also native-born. The deaths were of native-born, *i.e.*, the same group as the parents of the births. All racial elements (white) are included in births and deaths, but all are Americans in the sense of nativity.

$$\text{Vital index } B = \frac{100\ (\text{Births of whites, both parents foreign})}{\text{Deaths of foreign-born whites}}$$

Here again both births and deaths come from an identical group, in so far as foreign birth is concerned. The births are children of foreigners in this country. The deaths are of foreigners in this country.

$$\text{Vital index } C = \frac{100\ (\text{Births of negroes})}{\text{Deaths of negroes}}$$

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[22] Rubin, M., A measure of civilization, *Jour. Roy. Stat. Soc.*, vol. 60, pp. 148–161, 1897.
[23] Pell, C. E., *The Law of Births and Deaths*, London (Unwin), 1921, pp. 192.

This needs no discussion.

$$\text{Vital index } D = \frac{100 \text{ (Births of whites)}}{\text{Deaths of whites}}$$

This is for comparison with C. Both C and D are true vital indices, in the sense that the parents of the births in the numerator are drawn from the same population group as the deaths in the denominator.

Unfortunately on the basis of present published official compilations of statistics these four are the only significant vital indices which can be drawn up. For any really deep understanding of what the biological effect is of racial fusion, and of a new environment, on the net vitality of populations we ought to have a whole series of racially specific vital indices. Here again there is no practical hope of getting these from purely official sources. Some one must come forward and finance a comprehensive and thorough investigation along these lines from outside.

The facts about indices A, B, C, and D are set forth in table 62. In this table a figure in *italics* indicates that the absolute number of births and deaths on which the index is based is in each case less than 100. It will be noted that there are few such cases, and that they are practically all among the negroes of the northern states.

This table presents many novel points of great interest. We may first compare vital indices A and B, which indicate the relative biological vigor of the native-born and the foreign-born populations in this country. Taking totals first we note that for each grouping and each year index B is much larger than index A. Except for the rural population B is more than twice as large as A. Generally speaking the foreign population produces in this country approximately two babies for every death. The native population (as defined in vital index A) produces only a small fraction over one baby for each death. In other words the native population, even when so broadly defined as by index A, is in about the same state as France before the war, and not in as vigorous a state as the French population is now.

An examination of the same point by states is of interest. In Connecticut, in all but the influenza year 1918, index B was approximately four times as large as index A, and in 1918 it was three times. In this state the native population, even when so broadly defined as to involve necessarily only one full generation in this country, did not, in any of the four years reviewed come within a long distance of reproducing itself. It produced only about 0.8 of a birth for each death. Biologically that situation can have but one end.

TABLE 62

Vital indices of various elements in the population of registration states, cities in registration states, and rural portions of the registration states in the Birth Registration Area (1915–18 inclusive)

STATE AND GROUP		1915—VITAL INDEX				1916—VITAL INDEX				1917—VITAL INDEX				1918—VITAL INDEX			
		A	*B*	*C*	*D*	*A*	*B*	*C*	*D*	*A*	*B*	*C*	*D*	*A*	*B*	*C*	*D*
Connecticut	Cities	82.9	355.8	94.8	195.4	81.8	340.6	85.1	189.3	90.4	331.0	82.0	196.1	72.9	219.2	75.1	143.7
	Rural	75.7	292.0	*60.4*	149.0	73.5	282.9	*81.3*	146.4	77.8	293.2	*82.7*	149.2	65.7	212.2	65.0	120.2
	Total	80.5	339.7	86.0	180.9	79.2	326.6	84.3	176.7	86.4	322.6	82.1	182.8	70.8	217.7	73.4	137.3
District of Columbia	Total	117.0	97.5	85.2	123.0	119.7	93.5	87.3	125.9	126.6	103.7	85.5	131.7	102.1	78.5	64.7	104.2
Indiana	Cities	*	*	*	*	*	*	*	*	144.0	174.5	71.5	158.2	124.1	142.5	66.3	134.9
	Rural	*	*	*	*	*	*	*	*	172.5	54.3	59.1	166.3	153.2	53.1	53.8	149.0
	Total	*	*	*	*	*	*	*	*	162.7	121.8	68.2	163.3	142.8	108.5	63.3	143.6
Kansas	Cities	*	*	*	*	*	*	*	*	149.8	103.6	70.6	150.0	116.2	72.3	65.6	114.7
	Rural	*	*	*	*	*	*	*	*	223.7	58.1	74.5	208.2	190.4	50.5	67.4	177.9
	Total	*	*	*	*	*	*	*	*	207.3	68.6	72.2	195.3	171.9	50.1	66.4	162.2
Kentucky	Cities	*	*	*	*	*	*	*	*	135.8	26.6	47.4	123.1	105.6	25.1	34.7	98.5
	Rural	*	*	*	*	*	*	*	*	241.4	35.9	91.0	236.4	203.1	38.6	74.5	199.6
	Total	*	*	*	*	*	*	*	*	221.9	29.7	76.0	202.9	183.1	29.3	60.3	177.0
Maine	Cities	75.6	188.6	*71.4*	131.5	73.8	163.8	*25.0*	122.4	79.4	173.8	*75.0*	128.4	70.7	124.9	*53.8*	106.2
	Rural	105.6	156.0	*87.5*	136.4	105.7	146.2	*18.7*	135.9	115.0	161.6	*8.3*	145.5	96.5	112.5	*21.1*	119.9
	Total	98.8	169.0	*80.0*	135.1	98.1	151.3	*21.9*	132.3	106.3	166.7	*35.0*	143.0	90.2	117.8	*34.4*	116.2
Maryland	Cities	*	*	*	*	138.3	166.4	82.9	136.8	137.7	152.1	80.8	152.7	96.2	107.7	63.7	105.9
	Rural	*	*	*	*	177.0	90.1	128.6	173.5	173.3	82.6	125.1	168.7	129.8	67.6	92.8	126.3
	Total	*	*	*	*	157.5	144.9	106.8	164.6	155.1	132.2	103.1	160.1	111.9	96.8	78.8	114.9
Massachusetts	Cities	86.7	276.2	113.2	186.4	87.0	251.2	101.1	176.3	92.1	246.5	111.3	179.7	70.1	171.5	97.0	129.6
	Rural	80.5	226.4	70.6	145.1	79.0	202.1	73.5	135.3	77.4	207.4	128.4	135.6	63.0	147.8	86.2	104.8
	Total	85.1	267.1	105.8	177.0	85.1	242.3	96.0	167.2	88.5	239.6	113.3	169.9	68.5	167.4	95.4	124.5

Michigan	Cities	104.5	234.5	86.3	205.4	138.0	227.2	66.7	195.7	143.0	226.4	79.1	198.5	132.3	192.6	86.8	179.2
	Rural	182.2	143.5	83.1	197.9	172.2	140.5	68.1	186.7	171.7	139.4	64.1	185.3	155.9	123.8	77.0	167.4
	Total	165.0	187.8	85.2	201.2	157.2	184.4	67.1	190.9	158.8	184.2	74.5	191.5	145.2	159.7	84.1	173.0
Minnesota	Cities	173.0	166.3	59.9	211.5	163.2	148.2	51.4	194.5	170.8	136.3	67.7	194.5	137.0	105.1	61.1	156.4
	Rural	282.0	118.1	*18.1*	264.2	277.3	104.6	*88.2*	252.8	289.9	96.7	*50.0*	254.9	208.0	79.3	*42.9*	195.1
	Total	240.7	134.9	51.8	244.9	232.1	120.2	55.3	230.5	242.1	111.2	66.1	231.5	181.3	88.7	59.2	180.7
New Hampshire	Cities	70.3	293.7	*150.0*	163.0	72.4	248.4	*200.0*	153.9	69.2	239.5	*114.3*	147.1	60.9	164.1	*100.0*	113.7
	Rural	90.9	172.0	*50.0*	124.7	90.0	150.3	*30.0*	121.3	87.4	133.3	*60.0*	113.3	69.7	101.0	*20.0*	89.3
	Total	82.8	240.9	*110.0*	140.9	83.1	206.7	*69.2*	135.4	79.8	195.2	*91.7*	128.4	65.9	139.3	*71.4*	100.7
New York	Cities	88.4	273.5	94.9	179.5	88.5	255.4	101.6	172.5	95.6	246.0	96.3	175.2	79.4	187.1	84.5	137.1
	Rural	109.6	140.7	85.3	128.1	107.4	138.5	79.7	125.6	105.1	128.8	69.9	121.4	88.5	106.6	54.8	101.2
	Total	95.8	253.6	93.6	166.5	94.2	238.2	98.5	160.8	98.5	228.7	92.6	161.8	82.1	176.3	80.8	128.4
North Carolina	Cities	*	*	*	*	*	*	*	*	148.0	*60.9*	78.1	147.5	99.1	*32.9*	62.2	98.7
	Rural	*	*	*	*	*	*	*	*	266.8	*160.3*	190.3	270.4	223.7	47.0	145.0	224.4
	Total	*	*	*	*	*	*	*	*	255.4	106.3	173.1	258.2	209.2	41.6	33.8	209.3
Ohio	Cities	*	*	*	*	*	*	*	*	136.1	210.5	64.6	167.1	117.2	160.2	66.1	39.3
	Rural	*	*	*	*	*	*	*	*	157.5	113.3	73.8	156.3	138.2	98.5	70.1	137.5
	Total	*	*	*	*	*	*	*	*	147.0	182.2	67.2	162.0	127.7	143.3	67.2	138.5
Pennsylvania	Cities	117.2	273.3	95.2	179.2	110.4	253.8	87.6	166.5	114.6	243.1	74.4	166.3	81.1	153.7	59.3	112.5
	Rural	152.5	385.7	87.4	207.6	141.8	353.0	80.7	191.0	146.7	332.5	76.0	192.9	104.0	174.2	56.4	128.3
	Total	135.7	314.9	93.3	193.1	126.7	290.4	86.0	178.4	130.9	276.3	74.7	179.0	92.6	162.2	58.6	120.0
Rhode Island	Cities	74.3	219.5	83.2	158.3	72.5	216.4	76.1	152.7	79.2	217.8	89.5	159.2	64.0	172.5	91.6	126.0
	Rural	70.5	319.4	*65.8*	157.4	79.7	331.9	*113.6*	175.2	84.0	358.7	*123.8*	182.9	63.8	207.5	*29.8*	127.7
	Total	73.5	232.2	80.6	158.2	73.9	231.2	78.9	156.5	80.1	234.8	91.9	163.1	64.0	117.8	83.7	126.3
Utah	Cities	*	*	*	*	*	*	*	*	244.3	91.2	*140.0*	227.3	186.0	79.4	*75.0*	181.0
	Rural	*	*	*	*	*	*	*	*	390.7	97.1	*33.3*	338.9	312.7	71.6	*66.7*	274.2
	Total	*	*	*	*	*	*	*	*	339.4	94.7	*81.8*	297.8	265.4	74.8	*72.4*	238.5

* Not in the Birth Registration Area in designated year.

TABLE 62—*Continued*

STATE AND GROUP		1915—VITAL INDEX				1916—VITAL INDEX				1917—VITAL INDEX				1918—VITAL INDEX			
		A	*B*	*C*	*D*	*A*	*B*	*C*	*D*	*A*	*B*	*C*	*D*	*A*	*B*	*C*	*D*
Vermont	Cities	109.3	158.4	*100.1*	147.9	109.4	153.0	*100.0*	147.5	114.7	147.8	*100.0*	147.3	92.7	72.1	—	104.7
	Rural	121.3	138.4	*133.3*	147 2	110.7	135.5	*75.0*	134.8	114.3	134.7	*125.0*	136.6	96.5	91.3	*100.0*	111.5
	Total	119.9	142.5	*120.0*	147.3	110.5	139.0	*77.7*	136.7	114.4	137.3	*116.7*	138.2	95.9	86.5	*66.7*	110.4
Virginia	Cities	*	*	*	*	*	*	*	*	163.1	163.6	91.6	170.7	115.6	101.5	71.6	117.4
	Rural	*	*	*	*	*	*	*	*	255.3	125.4	159.2	252.6	200.7	103.6	137.0	200.1
	Total	*	*	*	*	*	*	*	*	233.4	144.7	139.2	232.6	177.7	102.4	117.1	176.8
Washington	Cities	*	*	*	*	*	*	*	*	169.1	123.2	*63.0*	184.8	132.2	84.3	*57.6*	140.3
	Rural	*	*	*	*	*	*	*	*	201.5	116.2	*42.3*	203.8	168.4	91.7	*56.8*	168.0
	Total	*	*	*	*	*	*	*	*	286.6	119.9	58.5	194.8	150.1	87.5	57.4	153.6
Wisconsin	Cities	*	*	*	*	*	*	*	*	178.4	142.2	75.0	194.8	143.2	115.9	*68.2*	156.9
	Rural	*	*	*	*	*	*	*	*	266.0	57.6	37.1	209.1	217.9	57.5	*65.2*	186.9
	Total	*	*	*	*	*	*	*	*	231.5	89.8	60.4	203.6	187.9	81.6	67.2	174.8
Totals	Cities	100.5	267.5	93.1	181.7	100.5	247.8	89.2	172.5	117.0	228.3	79.6	173.0	93.2	166.9	66.8	132.0
	Rural	141.1	215.4	82.5	179.0	137.7	199.9	109.0	170.1	177.7	156.5	146.2	187.4	144.8	118.8	118.4	150.8
	Total	117.8	252.4	91.4	180.7	116.3	234.1	94.2	171.6	148.1	205.2	114.3	179.8	118.8	151.8	93.7	140.6

The District of Columbia is in many respects demographically unique. Its abnormality is well reflected in the vital indices. *A*, while absolutely small, is still larger than *B* in each of the four years. The foreign population of the District succeeded in reproducing itself in only one of the four years, 1917, and then only barely so. Because of the obvious peculiarities of the population of the District no particular significance attaches to the result either way.

In Indiana, Kansas and Kentucky, in the two years for which data are available, the native population exhibits greater vitality than the foreign. (Index *A* greater than *B*) The only exception is in Indiana cities, where the usual relation of *B* greater than *A* prevails. The foreign population makes a particularly bad showing in the rural portions of these states. It is by no means wholly a question of smaller numbers involved. For example in 1917, 4066 foreign whites died in Indiana, and only about 2000 more (exactly 6305) died in Connecticut. But whereas there were but 4952 births of which both parents were foreign in Indiana, there were 20,341 such births in Connecticut. The Indiana foreign population, in respect of racial stamina, age distribution and otherwise, is a less fit and vigorous population than the Connecticut foreign population. What is of more interest is the comparison of vital index *A* for these three middle western states with *A* for Connecticut. We shall return later to a more particular discussion of this, merely noting now that index *A* is much higher in the middle west native population.

Maine is like Connecticut in the general trend of its figures for index *A* and index *B*. So also are the other New England states, Massachusetts, New Hampshire, and Rhode Island, with differences in degree in the several states. Vermont is the only New England state having index *A* generally larger than 100.

In Maryland the native white population is reproducing itself less effectively in the cities than the foreign white, but much more effectively in the rural districts, making for the whole state a net balance in respect of biological vitality in favor of the native.

Michigan shows the same relation as Maryland as between urban and rural, but the net balance of the total, owing to the distribution of the population between urban and rural, is in favor of the foreign.

Minnesota, Utah, Washington, and Wisconsin exhibit in varying degrees the same phenomenon we have seen in Indiana, Kansas and Kentucky. The native population is reproducing itself more effectively than the foreign, both in urban and rural communities.

New York, Ohio and Pennsylvania behave in general like the New England states, the foreign populations in varying degree exceeding the native in net vitality.

North Carolina and Virginia, the only southern states included in table 62, have small foreign populations, especially the former. The native population is on the whole definitely more effective in net vitality than the foreign.

Summarizing, we may say that in the New England states (with the exception of Vermont) and New York the native population is not reproducing itself, and in Vermont only barely so. The native rural population of New York State is just about breaking even between births and deaths. The foreign population is practically everywhere showing a higher degree of vitality except in Kansas, Kentucky, Utah and Wisconsin. Also in the rural portions of Indiana and Maryland their vital index falls below 100. The general facts leaving out the influenza year 1918 are summarized in table 63.

The relation between urban and rural population in respect of the vital index is interesting. So far as the white population is concerned, we note, having regard for indices A and B, that the following rules hold generally. The native population has a *lower* vital index in cities than in rural districts: the foreign population shows the reverse relation, the *higher* index being for the city populations. By and large, and with all factors included, as they are in the vital index, it appears that the foreign population, as it is actually constituted in the B. R. A. in respect of age, etc., is a biologically fitter population in the cities than is the native; while the native population is better under rural conditions.

A point might well be made here by a critic of the preceding results. He might say:

> It is all very well to show that the foreign population, as actually constituted in this country, shows a higher vital index than the native, but this fact may only mean that the foreign population is more favorably distributed in respect of age than the native, both in regard to deaths and to reproduction. If the foreign population contains a great many more people than the native population in early adult life, which is the period of a higher reproduction (birth) rate and a low death rate, all the results you have shown might well appear from this cause alone.

This is an entirely valid criticism so far as any evidence we have yet presented goes. The vital indices of table 62 are crude indices. We need age-specific vital indices for native- and foreign-born populations.

TABLE 63

States generally having

A LESS THAN 100			*A* GREATER THAN 100			*B* LESS THAN 100			*B* GREATER THAN 100		
Urban	Rural	Total	Urban	Rural	Total	Urban	Rural	Total	Urban	Rural	Total
Conn.	Conn.	Conn.	D. C.	Ind.	D. C.	D. C.	Ind.	D. C.	Conn.	Conn.	Conn.
Maine	Mass.	Maine	Ind.	Kan.	Ind.	Ky.	Kan.	Kan.	Ind.	Maine	Ind.
Mass.	N. H.	Mass.	Kan.	Ky.	Kan.	N. C.	Ky.	Ky.	Kan.	Mass.	Maine
N. H.	R. I.	N. H.	Ky.	Maine	Ky.	Utah	Md.	Utah	Maine	Mich.	Md.
N. Y.		N. Y.	Md.	Md.	Md.		Utah	Wis.	Md.	Minn.	Mass.
R. I.		R. I.	Mich.	Mich.	Mich.		Wis.		Mass.	N. H.	Mich.
			Minn.	Minn.	Minn.				Mich.	N. Y.	Minn.
			N. C.	N. Y.	N. C.				Minn.	N. C.	N. H.
			Ohio	N. C.	Ohio				N. H.	Ohio	N. Y.
			Pa.	Ohio	Pa.				N. Y.	Pa.	N. C.
			Utah	Pa.	Utah				Ohio	R. I.	Ohio
			Vt.	Utah	Vt.				Pa.	Vt.	Pa.
			Va.	Vt.	Va.				R. I.	Va.	R. I.
			Wash.	Va.	Wash.				Vt.	Wash.	Vt.
			Wis.	Wash.	Wis.				Va.		Va.
				Wis.					Wash.		Wash.
									Wis.		

Let us put the matter in this way: Suppose that a gigantic corral were constructed with two compartments. Suppose that further there were put into one of these compartments, on a given date, all the native-born women aged 20 to 24 inclusive say, while into the other compartment were put all the foreign-born women in the country of the same ages. Suppose them all to be told that they were to stay there for one year, but that men could have free access to the corrals for purposes of reproduction. Finally suppose that similar corrals were constructed, and the women impounded in them, for each age group from say 10 to 14 at one extreme to 55 and over at the other extreme.

In any one compartment of any one corral during the year (*a*) some of the women would have babies, and (*b*) some of the women would die. If

TABLE 64

Age specific vital indices for native-born and foreign-born women in B. R. A., 1919

AGE	BIRTHS FROM MOTHERS BORN IN UNITED STATES	DEATHS OF NATIVE-BORN FEMALES	VITAL INDICES FOR NATIVE WOMEN	BIRTHS FROM FOREIGN-BORN MOTHERS	DEATHS OF FOREIGN-BORN FEMALES	VITAL INDICES FOR FOREIGN WOMEN
10–14	391	5,002	7.82	15	268	5.60
15–19	77,048	7,763	992.50	10,768	759	1418.71
20–24	258,876	11,854	2183.87	74,247	2,120	3502.22
25–29	250,548	13,189	1899.67	102,429	3,317	3088.00
30–34	166,777	11,813	1411.81	83,326	3,583	2325.59
35–39	101,638	10,603	958.58	56,414	3,723	1515.28
40–44	33,832	9,511	355.71	18,878	3,566	529.39
45–49	3,202	10,092	31.73	1,866	4,120	45.29
50–54	68	10,926	0.62	54	4,968	1.09
55 and over	26	96,919	0.03	13	47,478	0.02
Totals.....	892,406	187,672		348,010	73,902	

we kept statistical record of these events we could, at the end of the year, calculate the age specific vital index for each group of women. It would not be the general population vital index because no male deaths were included (and cannot be because of lack of published data). But it would be an age-specific vital index for the females as reproductive units.

The results of exactly such an experiment for the women of the B. R. A. in the year 1919 are shown in table 64.

The figures in table 64 show plainly enough that at every age between 15 and 54 inclusive the foreign-born women have higher *specific* vital indices than native-born women. How much so is shown graphically in figure 35.

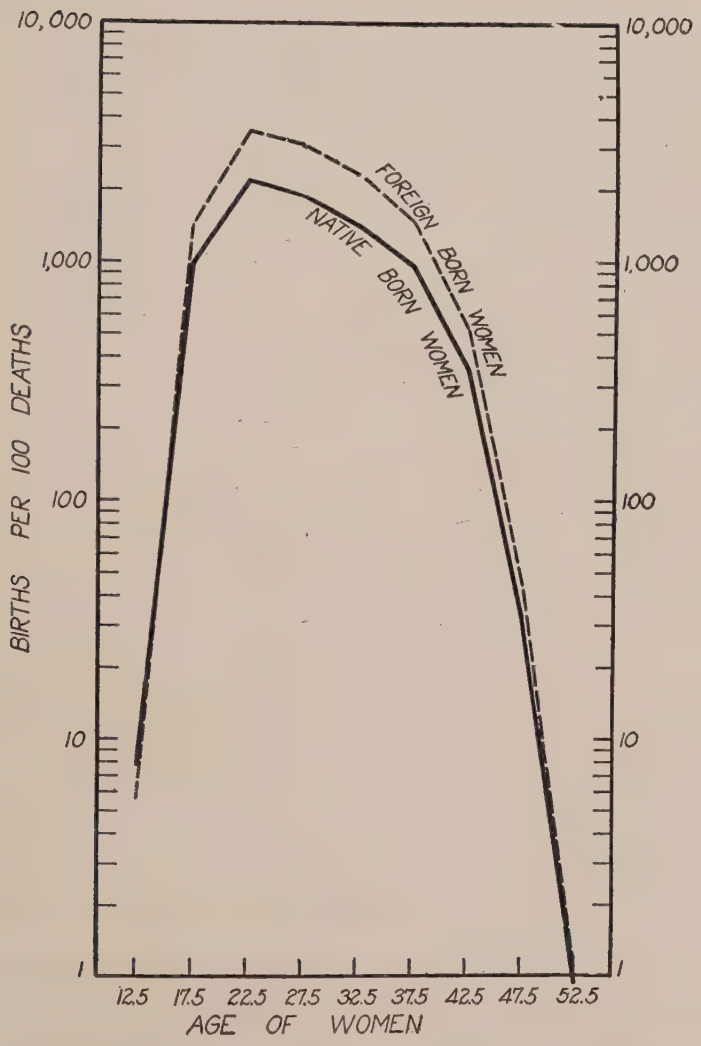

FIG. 35. SHOWING THE DIFFERENCES IN SPECIFIC VITAL INDICES FOR NATIVE-BORN AND FOREIGN-BORN WOMEN IN 1919

Solid line, native-born women; dash line, foreign-born women

As a reproductive machine the foreign-born woman far excels the native-born. For each native-born woman dying between 20 and 24 years of age, the native-born women as a group produce approximately 22 babies.

But for each foreign-born woman dying between 20 and 24, the foreign-born women as a whole produce 35 babies. It is in these five years that women, under conditions of life as now socially organized in the United States, do their best work biologically for the race, "best" being taken here in the sense of biological efficiency and economy.

So far as I am aware no attempt has been made hitherto to calculate age-specific vital indices. They picture, as exhibited in table 64 and figure 35, an extremely interesting biological fact. If we had such indices for populations of lower animals in different environmental situations, we should be in a position to know a great deal more than we now do as to the method of evolution. For it is the net balance between births and deaths which is the most significant information that can be had about the progress of the struggle for existence.

It may be objected in table 64 that we have put all births (both male and female) against only female deaths. The thought in doing this was that, after all, females have to produce *all* the babies whether the latter are boys or girls. If one wishes to postulate the problem in this way: how many new reproductive machines (females) do women of a specified age produce as a class for each similar reproductive machine lost by death, then of course one should take only female births in computing the specific vital indices. The result would be, of course, that the births and consequently the indices, table 64, would be about one-half as large absolutely as they are in that table, but the general *form* of the curve of figure 35 would be unchanged. What would be of great interest, but which we now unfortunately cannot do, is to make a table corresponding to table 64, but for *males* instead of females. Why we cannot do it is because the birth statistics published do not separate fathers by both age and nativity. We can, however, get fathers of all nativities by age, and from that see how the curve of age-specific vital indices runs for males in general, as compared with that for females.

The data for male age-specific vital indices are given for 1919 in table 65.

The striking differences between the vital indices of table 65 and those of table 64 are the higher values for males than females in the ages after 25 or 30, and the lower values earlier. The male specific vital index curve, as shown in figure 36, is flatter topped and more spread out in general than the female curves. This is what would be expected from the known facts about the male mortality and reproductive capacities relative to age.

With the data for specific vital indices in hand the obvious thing to do would be to proceed to the calculation, for each state and the B. R. A.

as a whole, of vital indices *corrected* for the age distributions of the several populations. Unfortunately, however, in this case as in so many others, we are balked in the performance of this desirable enterprise because the raw materials are not so tabulated in official publications as to make it possible. We shall have to resort to indirect methods to check up with more critical care the conclusions we have drawn from the crude vital indices in table 62.

The chief conclusions to which we have come so far from an examination of vital indices are these:

First, that generally index *B* (foreign population) is higher than index *A* (native population).

TABLE 65

Age specific vital indices for men in B. R. A., 1919 (births per 100 deaths)

AGES	BIRTHS	DEATHS	VITAL INDEX (BIRTHS PER 100 DEATHS)
10–14	15	7,086	0.21
15–19	14,211	11,395	124.71
20–24	190,061	14,804	1,283.85
25–29	338,605	17,685	1,914.65
30–34	313,294	19,136	1,637.20
35–39	239,578	20,401	1,174.34
40–44	131,025	18,666	701.94
45–49	65,728	20,807	315.89
50–54	20,627	22,032	93.62
55 and over	10,123	163,447	6.19
Totals.....	1,323,267	315,459	

Second, that generally index *A* is higher for rural than for urban populations.

Third, that generally index *B* is higher for urban than for rural populations.

We have now to ask this question: are these differences such in direction and magnitude as might reasonably be expected to arise merely from differences in the age constitutions of the several populations involved?

As the first step in answering this question table 66 is presented. This shows for each state in the B. R. A., in 1917 and 1918, the percentage of women of ages 15 to 44 inclusive in the following divisions of the population in 1910: (*a*) urban native-born white, (*b*) rural native-born white, (*c*) urban foreign-born white, (*d*) rural foreign-born white. The ages 15 to 44 were chosen because table 64 shows that it is only within these years that the female age-specific vital indices rise to values greater than 100.

From this table it is seen that, with some few exceptions, the following relations generally hold: The most favorable population in respect of the age distribution relative to vital index of females is the foreign urban, next comes the native urban, next the foreign rural, and last the native rural.

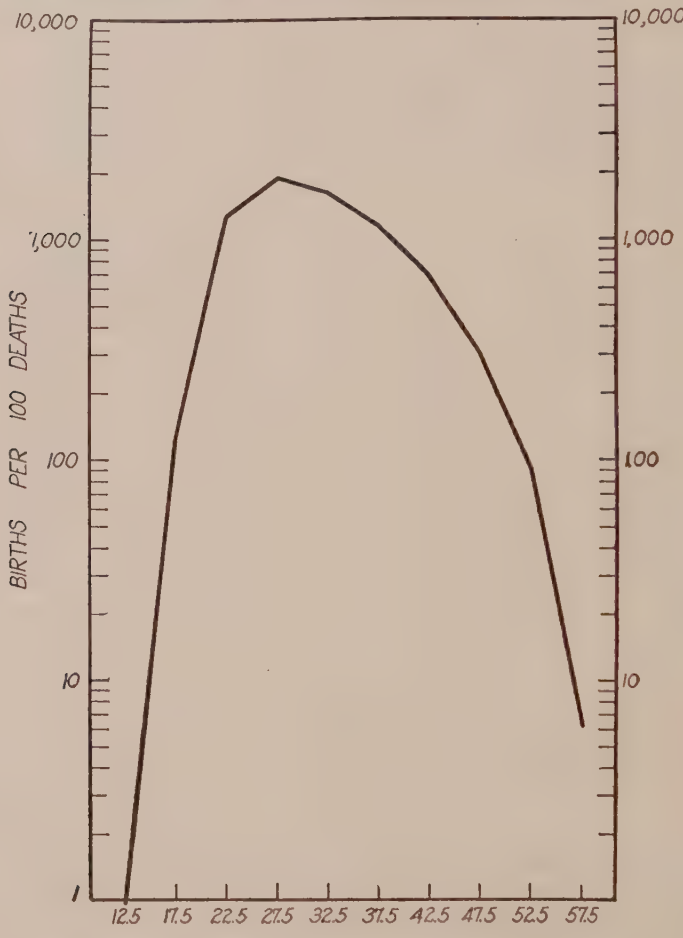

Fig. 36. Age-specific Vital Indices for Males (See Text)

Turning now to our first conclusion we have the following question: if the proportion of females 15 to 44 were the same in the native-born population that it is in the foreign-born population would the former population, in any of its divisions, show as high values for the vital index as the

latter population actually exhibits? The answer at once is that it could not, because table 64 shows that in each age class between 15 and 45 foreign-born women have a higher specific vital index than do native-born women, and if these indices were multiplied by the same population numbers the latter group would be bound to show a lower average vital index as a group. In other words, the foreign-born population has *both* a more favorable age distribution for the vital index, and also higher specific indices than the native-born.

TABLE 66

Percentage of women 15–44 years of age in the population in 1910

STATE	ORDER	NATIVE URBAN	ORDER	NATIVE RURAL	ORDER	FOREIGN URBAN	ORDER	FOREIGN RURAL
Connecticut	19	45.0	20	38.8	2	64.7	4	58.1
District of Columbia	4	52.7			15	54.5		
Indiana	6	52.4	5	45.3	17	49.2	20	31.4
Kansas	7	52.3	1	46.3	20	46.6	16	40.6
Kentucky	3	53.8	6	44.4	21	34.2	19	32.0
Maine	20	44.9	16	40.9	8	61.3	6	56.0
Maryland	12	50.8	4	45.5	16	54.0	12	46.5
Massachusetts	18	45.3	19	40.1	3	64.4	3	58.8
Michigan	11	51.1	11	43.7	13	55.4	13	44.1
Minnesota	2	54.3	8	44.3	11	58.1	14	42.4
New Hampshire	21	42.9	18	40.3	4	63.4	5	57.2
New York	15	48.0	10	43.7	1	64.8	10	48.9
North Carolina	10	51.4	12	43.3	9	58.7	9	52.1
Ohio	8	52.2	7	44.4	14	55.0	15	41.9
Pennsylvania	14	48.6	14	42.1	7	62.3	2	63.8
Rhode Island	17	45.4	17	40.5	6	62.5	1	66.1
Utah	13	50.4	13	43.1	19	46.9	17	39.5
Vermont	16	46.3	15	41.6	12	56.0	11	47.1
Virginia	5	52.5	9	43.8	10	58.1	8	54.3
Washington	1	54.9	2	45.7	5	62.8	7	54.5
Wisconsin	9	52.0	3	45.6	18	48.6	18	36.6

The next question is: Is the higher value of index *A* for the rural native than the urban native population due to a more favorable age distribution of the former? As an aid to answering this question figure 37 is presented.

The figure shows, first, the percentage of native women in the age period 15 to 44 inclusive in the urban population, the states being arranged in descending order of magnitude of this percentage. Next is plotted the percentage of this class of women in the rural population. It will be seen that in general this line runs parallel to, and everywhere *below*, the first.

In other words, the age distribution of the rural population is, in every state included, distinctly *less* favorable to a high value of the vital index than is that of the urban native population.

Against these percentages taken from table 66 are plotted urban and rural values of vital index *A* for the same states, taken from table 62. The striking thing at once apparent is that, with the exception of the three states Vermont, Massachusetts, and Connecticut, the urban line lies everywhere *below* the rural, by an amount generally substantially in excess of that which separates the percentage lines. In other words, we see that

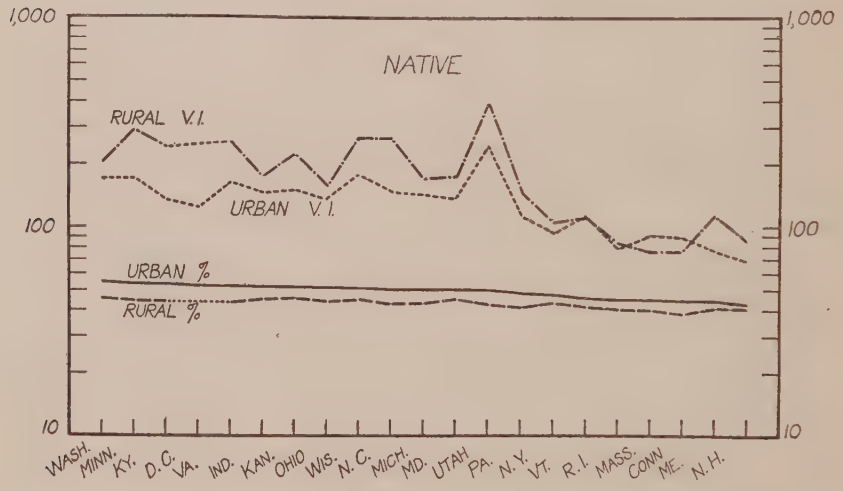

FIG. 37. SHOWING (*a*) PERCENTAGE OF NATIVE-BORN WOMEN 15 TO 44 IN URBAN POPULATION, (*b*) PERCENTAGE OF NATIVE-BORN WOMEN 15 TO 44 IN RURAL POPULATION, (*c*) VITAL INDEX *A* FOR URBAN POPULATION, (*d*) VITAL INDEX *A* FOR RURAL POPULATION

while the native urban percentage of females in the child-bearing ages is higher than the native rural percentage, and would therefore lead to the expectation of higher vital indices in the former population, actually the vital indices show the opposite relation. In view of these facts there can be no doubt of the justice of our conclusion that rural conditions are substantially more favorable to the biological vitality of the native population than are urban conditions. Or, put in another way, the native population appears to be better adapted biologically to country than to city life.

The data are plotted on a logarithmic scale in order that slopes of the lines may be comparable. It is seen that in general the four lines run,

in respect of their slopes, about parallel from the left margin of the diagram to and including Maryland. Utah's vital indices are much higher than would be expected from the age distribution of the child-bearing population alone. After that point clear to the right edge of the diagram, the relation between age distribution of the population and the values of the vital indices is obviously of a different order from that exhibited in the first half of the diagram. The New England states, for these are the ones here included, together with Pennsylvania and New York, have much lower vital indices for their native populations than the proportion of child-bearing women would entitle them to, on the basis of the showing

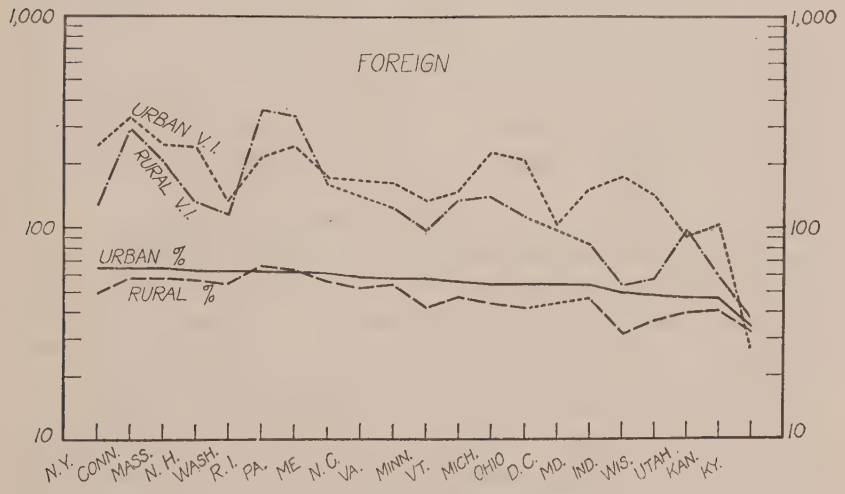

FIG. 38. SHOWING (*a*) PERCENTAGE OF FOREIGN-BORN WOMEN 15 TO 44 IN URBAN POPULATION, (*b*) PERCENTAGE OF FOREIGN-BORN WOMEN 15 TO 44 IN RURAL POPULATION, (*c*) VITAL INDEX *B* FOR URBAN POPULATION, (*d*) VITAL INDEX *B* FOR RURAL POPULATION

of the other states. This can only mean that in the New England states especially, and to a less degree in New York and Pennsylvania, the age-specific vital indices of the native-born women must be extremely low. The native populations of these states are in a biologically unhealthy condition.

The next question is: Is the general superiority of the foreign population in respect of the vital index in urban, as compared with rural environments, chiefly due to a more favorable age distribution of the child-bearing population in the former case?

Figure 38 puts the available data in graphic form.

The data plotted in this figure are the same, *mutatis mutandis*, as those in figure 37. The results, however, are quite different. Here the urban vital index line lies generally above the rural. In other words, it is apparent that a substantial part of the superiority of the foreign vital index under urban conditions must be in fact merely due to the more favorable age constitution of the urban as compared with the rural foreign population. Probably in most of the states nearly all of the superiority is due to this factor. It is, however, of interest to note that the vital index lines in figure 38 do not parallel throughout their course the age percentage lines, in respect of their slopes. The last 8 or so states at the right end of the diagram show generally lower vital indices, both urban and rural, than would be expected from the age constitution of the population, on the basis of the other states included in the diagram. These are middle western states for the most part, where the foreign-born population, while socially more desirable perhaps than the foreign-born population of the more highly industrialized eastern states, is nevertheless a biologically less vigorous population.

The question of the secular trend of the vital index of the several components of our population is of great interest, but could not in 1921 be satisfactorily dealt with for two reasons: first, because only four years were available and one of these was highly abnormal because of the influenza epidemic; and second because the B. R. A. changed its composition radically by addition in the middle of the period. In consequence of this latter point the totals for 1917 and 1918 were not comparable with those for 1915 and 1916. Now at the end of 1923 we have available three more years of statistics and this problem of trends of the vital indices is discussed in the next chapter of this book.

There is one point of interest in the general connection discussed here, namely, the effect of the influenza epidemic on the vital indices. Comparing the totals of 1918 with those of 1917 it appears that:

Vital index *A* (native white population) for cities fell 23.8 points or 20 per cent of its value in 1917

Vital index *A* (native white population) for rural areas fell 32.9 points or 19 per cent of its value in 1917

Vital index *A* (native white population) for total areas fell 29.3 points or 20 per cent of its value in 1917

Vital index *B* (foreign white population) for cities fell 61.4 points or 27 per cent of its value in 1917

Vital index *B* (foreign white population) for rural areas fell 37.7 points or 24 per cent of its value in 1917

Vital index *B* (foreign white population) for total areas fell 53.4 points or 26 per cent of its value in 1917

From these figures it is evident that the epidemic reduced the net vitality of the population from one-fifth to one-fourth of what it was in the preceding year. This was a sharp blow. Further it appears that rural populations were slightly, but probably not significantly, less damaged relatively, in a biological sense, than urban populations. The foreign population was definitely harder hit biologically than the native. This might reasonably have been expected from all that we know of the general hygienic ignorance of this element of the population.

Let us now turn to the examination of vital index *C* for the negro population in comparison especially with *D* (total white population) and *A* (native white). The material includes only three southern (or border) states with large negro populations, namely North Carolina, Virginia and Maryland. In a number of the northern states, however, the negro population is large enough to furnish births and deaths in sufficient magnitude to give reliable indices.

The immediate and striking thing which the indices exhibit in comparing *C* and *D* is the low value of the former compared with the latter in the years 1915 to 1918. Indeed one may say generally that, except in the rural districts of the southern states, practically never did the vital index of the negro population rise in those years to a value of as much as 100. Nowhere in *cities*, even in the southern cities, did the value of the negro vital index get to 100 in a fairly normal year such as 1917 except that the absolutely small negro population of Massachusetts cities did show an index slightly over 100.

Even in rural portions of the B. R. A. the negro index does not approach in magnitude the total white index nor the native white index for the same communities. This is true in the three southern states as well as in northern. These results suggest that the negro is biologically a less fit animal, *in the American environment physical, social and general*, than the white. It is idle to argue that if the negro were given a better chance, in a broad environmental sense, he might exhibit a degree of biological fitness, as measured by the vital index, equal or superior to that of the whites. But it will be wise to reserve judgment on these vital indices for negroes until the additional data presented in the next chapter have been analyzed. It is probable that a part of the low values of the indices is accounted for by incomplete registration of negro births in the southern states.

It is of interest to examine the comparative effect of the influenza epidemic on negro and white populations. Taking totals as before for 1918 as compared with 1917 we have:

Vital index *C* (negro population) for cities fell 12.8 points or 16 per cent of its value in 1917

Vital index *C* (negro population) for rural areas fell 27.8 points or 19 per cent of its value in 1917

Vital index *C* (negro population) for total areas fell 20.6 points or 18 per cent of its value in 1917

Vital index *D* (total white population) for cities fell 41.0 points or 24 per cent of its value in 1917

Vital index *D* (total white population) for rural areas fell 36.0 points or 20 per cent of its value in 1917

Vital index *D* (total white population) for total areas fell 39.2 points or 22 per cent of its value in 1917

The figures show that biologically the negro population of the B. R. A. was apparently less severely damaged by the epidemic than the white population. The relation between urban and rural areas is exactly the reverse for negroes of what it is for whites. The rural negro population index was reduced more by the epidemic than the urban negro index, both absolutely and relatively.

VII. CONCLUSIONS

Out of the mass of statistical detail which has been presented in the preceding sections what sort of a general picture emerges? Have we answered, on the basis of vital statistics, our initial question as to what *kind* of a population this country is likely to harbor in the future, near or remote?

It must be regretfully stated that we have not been able to answer this question in any final or particularly satisfactory manner. I think, however, that it may fairly be said that we have shown that the responsibility for this failure is not primarily ours. It arises, as has been specifically pointed out in many places, because in the first instance the existing official information is not tabulated in such a way as to make possible many of the inquiries we should like to undertake and which are absolutely essential, if we are ever to have any deep understanding of our demography. In the second place, some information which is needed is not collected at all.

So far as the first of these difficulties is concerned, I am of the opinion that but one solution is practically feasible or possible. It is for some private foundation or group of individuals, who may be sufficiently interested, to come forward and finance an investigative organization for a period of years, to work in coöperation with the Census Bureau (a cooperation which the Census Bureau would probably gladly enter into).

What this organization would do would be, in the first place, to tabulate in various new ways the raw data as to births and deaths, and in the second place proceed, after the preliminary tabulations were finished, to an exhaustive biometrical analysis of the data. The project is, from the standpoint of expense, by no means an impossible one, and I can think of no investigation likely to have such far-reaching anthropologic, eugenic, social, and even perhaps economic consequences, as this would. It is as true today as it was a quarter of a century ago when the eminent statistician, Dr. G. B. Longstaff,[24] said in a meeting of the Royal Statistical Society, devoted to a discussion of the official statistics of the United States, that:

> As a theater of statistical investigation at the present time, no country in the world was so interesting as the United States, but with the imperfect tools at command they were unable to deal with a number of problems, quite unequalled elsewhere for magnitude and interest.

This is not the place to present detailed plans for such an organization as that suggested, whether for its budget or its specific problems. The most I can hope for is that the discussion set forth in these pages may excite sufficient interest in the whole matter to make it profitable to outline specific plans for a real investigation.

Incomplete as our picture is as compared with what we should like to have, it still is a fact, I think, that the material which has been presented in these pages does give a little clearer and more comprehensive view than we have hitherto had of present tendencies, working in that great struggling mass of more than one hundred millions of individuals, who collectively make up the soul and body of these United States. Let us endeavor to summarize briefly what our findings are.

In the first place our population has indubitably passed the point of inflection of its growth curve, and from now on we may confidently expect an ever-increasing pressure of population on the means of subsistence, to use Malthus's classical phrase. The vital question is what kind of people are to inherit the blessings which Providence has so lavishly bestowed upon our portion of the North American continent? All cannot have them. There is not room enough. Man still breeds so fast that at all too frequent intervals he resorts to force to determine whether his or some other particular kind of people are going to enjoy the pleasures of continued existence.

[24] *Jour. Roy. Stat. Soc.*, vol. 57, p. 687, 1894.

The struggle may be varied in its manner. Economic pressure may in the long run be as effective an eliminant as machine guns. In general, unless forcibly prevented—which means finally by murder and sudden death—that people will presumably inherit the earth and the fullness thereof which has habitually the highest vital index. The advocate of birth control as a solution of the problem of population should remember this, and draw from it the logical conclusion that if, for any reason whatever, he does not want the people who have the highest vital index to be the inheritors, he must be prepared to do something a good deal more potent than merely to control the birth rate of *his* own kind of people which is, in practical effect, about all that he has done so far. And he must not forget that people who have a high vital index are apt as a group to be pretty good fighters, in a technical military sense.

In the United States at the present moment certain broad facts seem to be outstanding in their significance for the future. They are:

1. That newly arrived foreigners rather speedily fuse effectively with the stocks already here, to a degree much greater than is presupposed, at least in most popular discussions of the subject of immigration and related matters. The temperature of the melting pot is higher than we generally give it credit for being. It really melts to a definite and significant degree.

2. That immigrant stocks are in the stark, raw business of reproduction and evolutionary survival superior to native stocks, even to native stocks which have been here only a generation or two. Beyond this point there is no definite evidence, but all that can be inferred from the meager data available seems to indicate that it is a general rule that the farther we get in the fusion process from pure fresh immigrant stock the lower the vital index becomes. The end of the series is represented by the native stock of New England which is simply passing away, with considerable rapidity.

3. That native stock does better, in a biological sense, under rural than under urban conditions, while the opposite is true for immigrant stock.

From the standpoint of *quality* of the population in respect of health, intelligence, and all round efficiency, it is suggested that a wise policy from now on for the country to pursue would be one which might be called intermittent or periodic immigration. Let all immigration of any sort whatever be rigidly and completely prohibited for a period of say twenty years, to be followed by a period of say ten years of free immigration, with only such restrictions physical, economic and criminal, as we have in the immediate past imposed. During the period of no immigration

the foreigners of the last influx and their children would become sensibly assimilated and fused into the American population. For the most part they would have become Americans in a real sense of the word. Such a plan would further provide for that periodic inflow of new blood, of low living standards, which seems to be both an economic and social necessity, at least to civilizations which are still in the stage of industrial development and exploitation. Finally such a plan would, so far as one can foresee, postpone the period of pressing over-population as long as it can humanly be postponed, without the necessity of resorting to internal interferences of dubious practicability.

The United States has been, from its beginning, a gigantic experiment in human genetics. The experiment has gone on now three centuries, roughly speaking. When I compare the net result, as indicated by the human product now here—the product counted in the 1920 census—with the somewhat more nearly pure-bred peoples of Europe, I cannot force myself to take that pessimistic outlook that I am told by some of my "100 per cent Nordic" friends that I ought to. It seems to me, looking at the matter as a biologist, that a real, distinct, unique *American* people has evolved in the course of the experiment and is still continuing to evolve. Further, as people go, it is not a bad lot. Its most interesting and valuable feature is that it is still changing and evolving. I can find no manner of bitter feeling or even regret that the pure English-Scotch-Welsh stock of the original settlers will eventually not be the dominant element in the complex of American germ-plasms that it has been in the past, though personally I am wholly of that stock, and am, for three centuries, an American. Such a state of affairs was, in the natural course of events, bound to come about. There cannot, by any possibility whatever, be anything approaching biologically pure race stocks in this country a century hence. There are practically none now, with the exception of the Jews, and there is every present reason to believe that even they will be far less pure in 2021 than they are in 1921. How far we have progressed in only about one generation, in point of time, in racial fusion, is indicated by the comparison of a careful estimate of the racial stocks in the country in 1872 with conditions now existing. In the *Journal of the Royal Statistical Society* in 1872,[25] it was estimated that the following figures gave the percentages of the different race stocks in the population at that time:

[25] The pedigree of the United States, *Jour. Roy. Stat. Soc.*, vol. 35, pp. 541–543, 1872.

	Per cent in population
Original British Stock	46
Irish	16
German	13
African (negro)	12
Recent British	8
French, Spanish and others	5
	100

The kind of people who will survive and run the affairs of the country, say a couple of centuries hence, when population pressure will be intense, will, I think, not be Englishmen, or Slavs, or Jews, or Italians, but *Americans*, of that type which has shown the greatest adaptability to the problems which life in this part of North America has presented. I think they will probably be just as gentle, as high-minded, as clever, as honorable, and as independent as any people on the face of the earth.

VIII. SUMMARY

1. It is shown elsewhere in this book[26] that the population of the United States (*a*) is growing according to a curve the equation of which is known; (*b*) has passed the point of inflection of this curve; and (*c*) is rapidly approaching its asymptote.

2. The problem of the future is the *quality* of the asymptotic population. To estimate this from past conditions is one of the purposes of this study.

3. It is shown that there are three general criteria by which the biological attributes of a population may be estimated or measured. These are: (*a*) somatic physical, (*b*) somatic psychological, and (*c*) biostatistical. These are defined and discussed. This study deals solely with the third of these criteria, including marriages, divorces, births, deaths, and the birth/death ratio.

4. Racial assortative mating in marriages effective in producing offspring in 1919 is discussed.

5. For the entire B. R. A. the percentage of amalgamation or fusion of foreign-born stocks with native-born, as compared with racially like effective matings of all sorts, as indicated by effective marriages in 1919, is just under 11.5 per cent.

[26] *Cf.* Chapters XXIV and XXV.

6. The amount of racial amalgamation or fusion going on in the several states of the Registration Area is proportional in the most direct and close way to the amount of foreign-born white stock in the local population.

7. The same result holds if the percentage of fusion matings of foreign with native stock is taken upon the base of pure native matings, instead of all pure matings as before.

8. In ten out of twenty-three states there are *more* effective mixed matings of the amalgamating type (*i.e.*, foreign × native) than there are of racially true foreign × foreign matings.

9. In the three states Massachusetts, New York, and Connecticut, there were in 1919 as many or more matings of the type foreign-born × foreign-born as there were of the type native-born × native-born, *effective in producing offspring*. In these three states as large or larger additions to the future population were made in 1919 by the foreign-born as by the native-born. Further, in two other states, New Hampshire and Pennsylvania, the effective foreign × foreign matings were more than half as numerous as the effective native × native.

10. The foreigner in this country is much more apt to marry an American-born person, if he does not marry one of his own race, than he is to marry some other foreigner not of his own race.

11. In 1919, there were in the B. R. A. 78 racially different types of mating effective in producing offspring, not separating sexes of either mated partners or offspring. Sixty-five per cent of the births were from native × native matings, 10 per cent of the children had one parent native-born and the other foreign-born, leaving approximately 25 per cent of the births having both parents foreign-born.

12. In those parts of the country where a relatively large proportion of the population is foreign-born, the fertility of the foreign-born women is greatly in excess of that of the native-born. Almost, if not quite, the first biological result of Americanization is to reduce the fertility of marriages.

13. The illegitimate rate is higher per 1000 foreign-born women capable of having an illegitimate baby than it is per 1000 native born women in the same social situation. But per 1000 total births the illegitimate rate is smaller for foreign- than for native-born mothers. The explanation of this apparent paradox is given.

14. The stillbirth rate is higher, on the basis of total live births, for foreign- than for American-born women in general. In the case of Scandinavian, German, Polish and Hungarian mothers, the stillbirth rate is lower than for native-born mothers.

15. After infancy and early childhood the age- and sex-specific death rates are lower at all ages for native-born of native parents than for either native-born of foreign parents, or foreign-born. A detailed discussion of mortality and race stock is presented.

16. The biological vigor as indicated by the vital index (100 births/deaths) of the native-born population is much lower than that of the foreign-born population. In the New England states (except Vermont) and New York, the native population is not reproducing itself, and in Vermont only barely so.

17. The native population has a lower vital index in cities than in rural districts; the foreign population shows the reverse relation, the higher index being for the city population.

18. A study of age-specific vital indices for women shows that as a reproductive machine the foreign-born woman far excels the native-born. For each native-born woman dying between 20 and 24 years of age, the native-born women as a group produce approximately 22 babies. The corresponding figure for foreign-born women is 35.

19. The peak value of the vital index for women falls in the age group 20 to 24, inclusive. For men the peak value is in the period 25 to 29.

20. Age and sex corrected vital indices are discussed.

21. Rural populations were slightly, but probably not significantly, less damaged relatively, in a biological sense, than urban populations by the influenza pandemic. The foreign population was definitely harder hit biologically by the pandemic than the native.

22. Except in the rural districts of the southern states the vital index of a negro population did not, except in a few instances, rise to a value of as much as 100 during the years covered in this study. Practically nowhere in cities, even in southern cities, does the value of the negro vital index get to 100.

23. Biologically the negro population of the B. R. A. was distinctly less severely damaged by the influenza epidemic than the white population.

CHAPTER IX

Trends of Vital Indices[1]

It has been shown in earlier papers[2] that the growth of human populations, within a given cultural epoch, is described with remarkable accuracy by a curve of the same sort mathematically as that which describes the growth in size of an individual organism, and also certain sorts of chemical reactions. Inasmuch, however, as this same type of curve also describes equally well other sets of phenomena which can possibly have only the most remote, if any, relation to any biological phenomenon, it seems to me evident that the mere fact that a particular sort of equation describes population growth can give but little if any insight into the operation of the biological factors which underlie this growth. The chief usefulness of the mathematical expression of the facts appears at present to flow from two considerations. The first of these is that by the use of the equations we can, within certain limitations, predict future population growth by extrapolation. The second is that we can smooth observed records of population growth, and by so doing get a result probably more nearly true than the actual census count. It is in these directions only that use has been made of the theoretical curves of population growth in this laboratory. A complete discussion of the significance and use of these curves is given in Chapters XXIV and XXV of this book.

The actual social problem of population is a much more vital and human matter than the graduation of census counts by a nicely adjusted mathematical theory. It concerns us personally, not merely academically. Specifically we here in the United States want to know how we may most intelligently go about safeguarding the quality of our future population. How much and in what way shall immigration be restricted, for example? Can anything be done to stay the dying out of old American stock? Are

[1] The paper on which this chapter is based was read before the American Sociological Society on December 28, 1923. It is here published for the first time.

[2] Pearl, R., and Reed, L. J., On the rate of growth of the population of the United States since 1790 and its mathematical representation, *Proc. Nat. Acad. Sci.*, vol. 6, pp. 275–288, 1921; A further note on the mathematical theory of population growth, *ibid.*, vol. 8, pp. 365–368, 1922; *Predicted Growth of Population of New York and Its Environs*, New York, 1923, pp. 42; On the mathematical theory of population growth, *Metron*, vol. 3, pp. 6–19. 1923. See Chapters XXIV and XXV of this book.

the farmers of the future going to be of American stock, or people of more recent foreign origin, satisfied with a lower standard of living than our people of longer residence here? And so on.

Fundamentally these are biological questions. Some of them I[3] discussed at some length in 1921. In that discussion the most significant biostatistical constant from the point of view of getting at the biological essentials of population questions appeared to be the "vital index" or birth/death ratio of a population. This is evident *a priori*. The basic biological elements in determining rate of natural population growth are clearly births and deaths. The ratio between these two phenomena *measures*, directly and simply, the natural increase of population. Furthermore Pearl and Burger[4] have shown with English statistics going over three-quarters of a century that there is, in that country at least, an extraordinarily perfect regulatory adjustment between births and deaths in a population, which keeps the ratio between them very nearly constant, despite first rising and then falling birth rates, and somewhat irregularly falling death rates. The behavior of the vital index in its quantitative aspects suggests an analogy to some of the internal regulatory phenomena of the living organism.

In Chapter VIII I dealt with four vital indices for the American population, described as follows (*loc. cit.*, pp. 228, 229):

$$\text{Vital index } A = \frac{100\ (\text{Births of whites of native parents})}{\text{Deaths of all native whites}}$$

$$\text{Vital index } B = \frac{100\ (\text{Births of whites, both parents foreign})}{\text{Deaths of foreign-born whites}}$$

$$\text{Vital index } C = \frac{100\ (\text{Births of negroes})}{\text{Deaths of negroes}}$$

$$\text{Vital index } D = \frac{100\ (\text{Births of whites})}{\text{Deaths of whites}}$$

Data for these four vital indices were given in table 62 of chapter VIII, for the four years 1915 to 1918 inclusive, those being the only years for which statistics were available when the first work was done.

[3] Pearl, R., The vitality of the peoples of America, *Amer. Jour. Hyg.*, vol. 1, pp. 592–674, 1921. See Chapter VIII of this book.

[4] Pearl, R., and Burger, M. H., The vital index of the population of England and Wales, 1838–1920, *Proc. Nat. Acad. Sci.*, vol. 8, pp. 71–76, 1922. See Chapter XXIII of this book.

On the basis of this material the following conclusions among others were drawn:

The biological vigor as indicated by the vital index (100 births/deaths) of the native-born population is much lower than that of the foreign-born population. In the New England states (except Vermont) and New York, the native population is not reproducing itself, and in Vermont only barely so.

The native population has a lower vital index in cities than in rural districts; the foreign population shows the reverse relation, the higher index being for the city population.

Rural populations were slightly, but probably not significantly, less damaged, relatively in a biological sense, than urban populations by the influenza pandemic. The foreign population was definitely harder hit biologically by the pandemic than the native.

Except in the rural districts of the southern states practically never does the vital index of a negro population rise to a value of as much as 100. Practically nowhere in cities, even in southern cities, does the value of the negro vital index get to 100.

Biologically the negro population of the B. R. A. was distinctly less severely damaged by the influenza epidemic than the white population.

In this earlier study it was stated (p. 244) that:

The question of the secular trend of the vital index of the several components of our population is of great interest, but cannot be satisfactorily dealt with for two reasons; first, because only four years are available and one of these is highly abnormal because of the influenza epidemic; and second because the B. R. A. changed its composition radically by addition in the middle of the period. In consequence of this latter point the totals for 1917 and 1918 are not comparable with those for 1915 and 1916. When data have accumulated for a larger number of comparable years it will be profitable to go into the problem in detail.

It is the purpose of the present chapter to discuss this question of trend, since we now have available statistics for three additional years, 1919 to 1921 inclusive, giving a total time base of seven years. This is not a long period to be sure, but perhaps we can get from its study some tentative suggestion as to the direction in which some of the elements of our population are moving.

Table 67, which is fashioned on exactly the same plan as table 62 of chapter VIII, gives the data for the years 1919, 1920, and 1921.[5] Indices printed in *italics* indicate that the ratio is based on fewer than 100 births or deaths.

We may first consider the trend of the vital index of the native-born as compared with the foreign-born elements in the population. Owing to the change in the composition of the Birth Registration Area during the period covered the total figures cannot be used. Instead attention will be confined to the original birth registration states. These were: Connecticut, District of Columbia, Maine, Massachusetts, Michigan,

[5] I am indebted to Dr. John Rice Miner for the calculation of table 67.

TABLE 67

Vital indices of various elements in the population of registration states, cities in registration states, and rural portions of registration states in the Birth Registration Area (1919–1921 inclusive.)

STATE AND GROUP		1919—VITAL INDEX				1920—VITAL INDEX				1921—VITAL INDEX			
		A	*B*	*C*	*D*	*A*	*B*	*C*	*D*	*A*	*B*	*C*	*D*
California	Cities	107.5	83.5	90.5	116.2	125.9	95.8	89.2	136.0	133.7	108.5	108.0	145.3
	Rural	109.4	104.6	63.2	120.0	124.5	123.5	71.4	139.3	132.1	138.7	79.6	148.4
	Total	108.3	91.5	83.0	117.8	125.4	106.3	84.3	137.3	133.0	120.2	100.1	146.6
Connecticut	Cities	99.9	331.9	117.6	202.5	104.4	303.5	115.7	195.4	128.7	346.4	147.6	231.2
	Rural	82.5	284.2	*91.4*	153.6	78.5	242.4	*93.8*	139.1	90.8	257.5	*112.1*	157.7
	Total	94.4	320.3	112.5	187.9	97.8	291.8	112.8	182.1	118.5	327.7	141.9	212.6
Delaware	Cities	*	*	*	*	*	*	*	*	154.6	301.3	113.8	209.0
	Rural	*	*	*	*	*	*	*	*	159.3	*163.1*	106.0	163.6
	Total	*	*	*	*	*	*	*	*	157.2	270.7	108.6	185.9
District of Columbia	Total	138.3	110.6	100.4	143.7	147.3	112.6	109.1	151.8	158.4	118.4	124.3	161.1
Indiana	Cities	150.2	181.3	93.1	165.7	157.0	164.4	91.2	168.2	187.8	190.5	123.1	200.2
	Rural	165.9	51.6	64.8	161.7	174.8	32.7	49.1	168.3	202.9	38.5	59.9	195.8
	Total	160.6	125.5	86.6	163.2	168.0	111.6	83.4	168.3	197.2	129.8	110.7	197.5
Kansas	Cities	151.2	124.1	77.9	154.4	173.2	91.6	89.2	167.8	203.7	113.6	106.2	201.2
	Rural	226.2	61.8	91.7	209.7	233.1	55.0	76.4	216.3	273.1	64.7	85.6	251.1
	Total	208.3	75.2	83.3	196.7	216.3	64.9	84.4	202.7	253.8	77.4	98.5	237.4
Kentucky	Cities	125.3	24.2	46.3	115.4	170.6	20.3	58.4	153.9	193.9	25.7	70.1	176.1
	Rural	240.2	55.7	101.3	235.2	275.5	43.4	108.0	270.3	331.5	70.4	125.1	327.4
	Total	217.1	33.7	81.1	208.7	254.7	27.6	90.7	244.7	302.8	38.9	105.7	292.6

Maine	Cities	82.5	171.5	*180.0*	131.1	95.7	145.1	*25.0*	137.3	105.3	183.4	*50.0*	158.8
	Rural	111.5	137.2	*30.8*	140.8	123.4	122.9	*13.3*	150.3	136.5	131.0	*30.0*	166.2
	Total	104.5	151.3	*95.7*	138.2	115.6	133.0	*19.4*	146.3	127.9	153.8	*38.9*	164.0
Maryland	Cities	153.2	158.1	107.3	166.6	167.7	152.8	111.3	179.7	182.7	154.1	129.0	193.0
	Rural	168.9	68.5	131.0	164.6	190.5	74.8	150.3	186.4	208.4	87.3	163.4	204.3
	Total	160.1	140.5	119.6	165.8	177.3	137.4	129.9	182.3	193.8	141.5	146.2	197.5
Massachusetts	Cities	96.3	237.1	101.5	179.0	107.6	220.9	124.5	181.5	127.6	234.3	144.5	205.7
	Rural	76.6	204.2	135.9	135.4	83.7	183.6	122.9	133.4	93.7	190.4	89.1	145.9
	Total	91.6	231.4	105.7	169.5	102.6	215.3	124.3	172.4	120.2	227.8	136.7	194.0
Michigan	Cities	148.2	200.1	111.9	195.0	150.0	197.9	91.3	193.0	191.1	238.2	165.0	243.1
	Rural	168.1	127.0	85.0	178.7	173.1	89.1	62.4	172.3	202.2	100.1	90.2	198.9
	Total	158.8	165.5	105.5	186.6	161.3	150.3	86.6	183.2	197.0	173.8	152.0	220.5
Minnesota	Cities	169.3	113.7	76.0	186.6	171.4	98.9	72.9	180.3	211.4	97.2	93.0	210.2
	Rural	251.5	80.3	*26.1*	223.9	301.1	69.4	*27.8*	248.0	356.5	75.8	*83.3*	286.4
	Total	220.6	92.2	69.2	210.2	242.7	80.8	68.6	219.2	291.7	84.4	92.1	253.8
Mississippi	Cities	*	*	*	*	*	*	*	*	190.9	*147.6*	73.6	189.0
	Rural	*	*	*	*	*	*	*	*	353.4	159.5	196.7	346.8
	Total	*	*	*	*	*	*	*	*	328.3	155.6	179.0	321.4
Nebraska	Cities	*	*	*	*	161.9	130.0	86.9	171.6	177.4	146.9	101.6	190.6
	Rural	*	*	*	*	315.9	51.1	*43.3*	274.3	359.0	55.8	*80.0*	308.1
	Total	*	*	*	*	269.3	75.8	81.1	242.8	302.4	83.0	99.5	271.8
New Hampshire	Cities	86.6	232.8	*180.0*	162.4	100.8	225.4	*11.1*	174.8	119.6	240.0	*225.0*	198.4
	Rural	87.6	129.4	*100.0*	114.0	103.7	120.6	*250.0*	126.1	115.0	130.4	*133.3*	141.1
	Total	87.2	189.3	*136.4*	135.0	102.5	179.0	*54.5*	147.2	116.9	193.3	*185.7*	166.0

* Not in Birth Registration Area in designated years.

TABLE 67—*Continued*

STATE AND GROUP		1919—VITAL INDEX				1920—VITAL INDEX				1921—VITAL INDEX			
		A	*B*	*C*	*D*	*A*	*B*	*C*	*D*	*A*	*B*	*C*	*D*
New Jersey	Cities	*	*	*	*	*	*	*	*	148.2	306.1	146.1	231.5
	Rural	*	*	*	*	*	*	*	*	132.7	216.4	128.7	172.4
	Total	*	*	*	*	*	*	*	*	142.3	280.3	140.5	210.6
New York	Cities	100.3	233.5	114.4	172.5	109.9	222.7	127.3	176.8	127.7	242.7	147.9	200.6
	Rural	105.8	122.0	59.5	120.4	110.6	117.0	70.0	124.5	124.6	133.0	72.9	141.1
	Total	101.9	217.5	107.5	159.7	110.1	207.7	121.7	164.0	126.7	227.8	139.7	185.4
North Carolina	Cities	147.0	*63.8*	90.1	147.0	191.9	*104.2*	103.7	193.0	229.8	*86.8*	130.3	230.7
	Rural	292.9	*83.1*	193.7	294.2	301.0	*88.9*	218.5	299.5	358.6	*120.3*	265.1	359.3
	Total	276.6	75.5	178.8	277.4	283.9	97.0	194.5	282.6	333.6	*102.4*	237.4	333.9
Ohio	Cities	136.1	196.8	90.8	166.2	152.8	184.1	96.8	177.3	175.9	214.1	124.5	204.9
	Rural	150.8	115.3	84.1	151.6	162.5	99.0	97.5	161.8	187.5	132.7	109.2	187.7
	Total	143.7	173.3	89.0	159.3	157.5	161.7	97.0	170.4	181.7	192.9	120.7	197.0
Oregon	Cities	127.6	77.7	*60.0*	133.4	145.1	79.5	*92.0*	150.2	164.8	81.1	*64.0*	166.8
	Rural	181.1	55.2	*18.2*	170.2	183.3	53.2	*62.5*	173.7	217.1	61.1	*8.3*	202.7
	Total	158.6	66.5	*48.8*	154.1	167.3	68.0	*84.8*	163.2	194.8	72.2	*45.9*	186.5
Pennsylvania	Cities	123.8	245.3	109.8	174.0	131.4	224.1	105.8	176.1	151.1	245.3	134.1	200.0
	Rural	147.0	330.9	107.4	192.2	159.1	301.4	110.7	198.2	181.6	354.4	113.0	227.3
	Total	135.8	277.0	109.2	182.9	145.1	250.7	106.8	186.4	166.6	283.0	129.3	213.0
Rhode Island	Cities	*	*	*	*	*	*	*	*	117.5	218.1	144.0	193.8
	Rural	*	*	*	*	*	*	*	*	98.5	209.8	*46.7*	162.1
	Total	*	*	*	*	*	*	*	*	114.0	217.1	129.8	188.5

South Carolina	Cities	163.2	*95.7*	78.7	159.6	162.4	*57.1*	80.1	157.8	197.2	*109.2*	94.2	197.5
	Rural	278.4	*54.3*	167.4	275.1	278.8	*82.5*	183.2	277.7	336.6	*89.7*	232.4	335.7
	Total	260.5	75.0	156.5	256.2	255.6	65.7	167.0	252.7	308.1	100.7	207.9	306.5
Utah	Cities	221.9	78.6	*84.2*	205.0	228.2	62.1	*85.7*	203.3	261.5	78.1	*63.6*	236.0
	Rural	353.5	77.1	*66.7*	310.4	372.6	81.5	*137.5*	331.1	405.5	89.9	*180.0*	362.6
	Total	303.7	77.8	*80.0*	268.0	311.9	71.9	*100.0*	274.0	346.4	84.2	*85.2*	307.7
Vermont	Cities	114.6	119.9	*0*	141.1	122.0	103.6	*0*	145.7	145.6	118.1	*0*	165.8
	Rural	118.8	122.3	*75.0*	137.9	114.0	112.2	*20.0*	131.9	137.1	134.5	*33.3*	157.6
	Total	118.2	121.8	*75.0*	138.3	115.1	110.6	*16.7*	133.9	138.2	131.1	*25.0*	158.8
Virginia	Cities	179.9	161.5	111.1	185.6	183.5	152.1	112.6	191.3	211.7	157.4	135.4	218.9
	Rural	240.8	108.7	162.9	239.3	266.5	111.0	197.4	266.9	301.9	164.1	216.6	303.6
	Total	226.0	134.8	147.6	225.8	244.7	132.4	169.1	246.4	279.5	160.2	191.2	281.8
Washington	Cities	161.3	101.1	*78.7*	170.3	169.4	89.6	*65.4*	172.6	190.8	99.3	*45.2*	196.4
	Rural	181.9	85.7	*48.0*	178.0	192.6	85.1	*53.8*	185.3	217.5	89.7	*34.1*	209.2
	Total	171.9	93.9	*69.8*	174.1	180.8	87.6	*62.2*	178.7	204.3	94.8	*41.8*	202.7
Wisconsin	Cities	178.2	123.4	*69.7*	188.2	183.3	108.3	*96.3*	185.9	228.7	112.8	*116.4*	220.7
	Rural	243.3	56.0	*30.3*	201.1	258.1	50.7	*27.8*	207.8	285.0	51.4	*80.8*	227.3
	Total	218.3	81.8	*56.6*	196.1	226.5	73.8	*75.0*	198.7	262.9	75.3	*104.9*	224.7
Totals	Cities	122.8	206.5	98.5	169.6	134.9	193.8	102.8	175.3	156.2	219.0	124.4	202.1
	Rural	175.7	141.4	157.4	182.8	191.6	123.7	177.2	194.1	219.1	145.7	204.1	222.1
	Total	150.1	184.9	133.3	175.8	163.0	171.3	143.3	183.9	187.9	195.5	171.6	211.3

TABLE 68

Vital indices of various elements in the populations of (a) the original birth registration states, and (b) a group of southern registration states

		1915				1916				1917			
		A	*B*	*C*	*D*	*A*	*B*	*C*	*D*	*A*	*B*	*C*	*D*
Original Birth Registration States	Cities	100.5	267.5	93.1	181.7	99.1	249.4	90.7	173.0	105.1	242.0	85.1	175.4
	Rural	141.1	215.4	82.5	179.0	135.6	201.9	78.7	169.9	137.9	196.1	76.3	170.1
	Total	117.8	252.4	91.4	180.7	114.4	235.8	88.8	171.8	118.8	229.2	83.8	173.4
Southern Birth Registration States	Cities	117.0	97.5	85.2	123.0	132.5	146.6	84.9	147.3	141.4	113.6	79.5	145.6
	Rural					177.0	90.1	128.6	173.5	244.2	79.8	158.6	241.5
	Total	117.0	97.5	85.2	123.0	150.5	134.1	100.9	157.1	216.9	103.9	132.4	213.5

		1918				1919				1920			
Original Birth Registration States	Cities	83.1	175.2	71.3	132.0	112.3	232.1	108.8	176.6	121.3	217.7	111.6	179.0
	Rural	108.6	138.0	59.0	129.8	137.7	182.8	93.8	166.7	148.5	159.0	96.1	170.8
	Total	93.6	164.5	69.3	131.1	123.0	218.1	106.5	172.8	132.2	201.8	109.5	176.0
Southern Birth Registration States	Cities	103.2	85.8	62.0	105.7	150.1	120.5	93.5	154.0	170.8	114.8	100.1	173.0
	Rural	198.9	66.3	127.9	196.5	251.3	74.7	165.3	248.3	272.7	75.0	187.5	269.9
	Total	170.2	80.5	106.3	167.0	223.3	108.9	145.9	219.9	242.6	105.0	160.9	239.1

		1921			
Original Birth Registration States	Cities	142.2	237.7	139.3	205.2
	Rural	169.3	182.4	100.1	194.5
	Total	153.4	222.8	133.4	201.2
Southern Birth Registration States	Cities	192.9	121.8	113.4	193.5
	Rural	323.6	106.0	216.6	321.2
	Total	286.4	118.0	188.7	281.8

Minnesota, New Hampshire, New York, Pennsylvania, Rhode Island and Vermont. During the two years 1919 and 1920 the figures for births in Rhode Island are omitted from the annual volumes of Birth Statistics, but because it made no significant difference in the results this state has been included in the totals for the available years. The vital indices

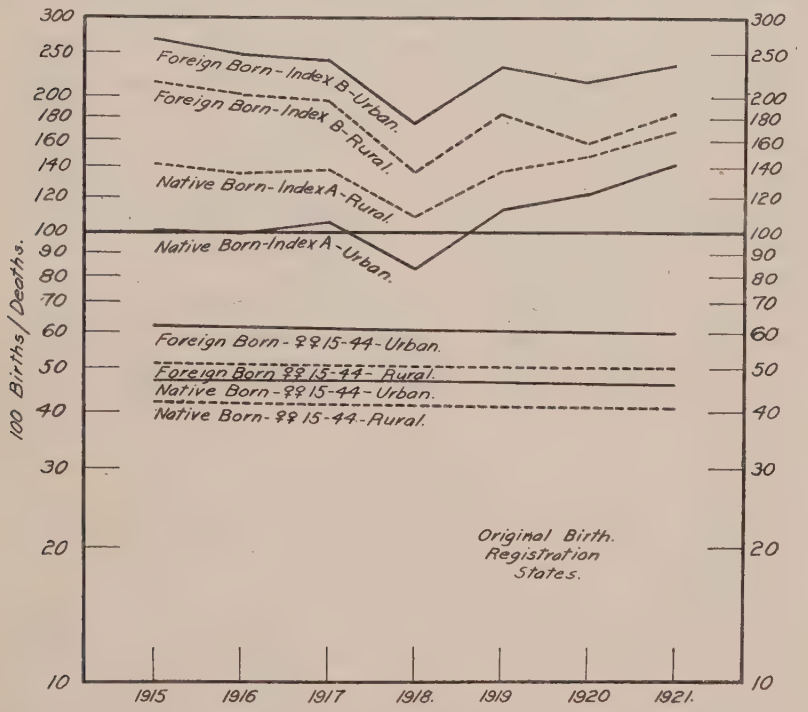

FIG. 39. THE COURSE OF VITAL INDICES *A* AND *B* (NATIVE-BORN AND FOREIGN-BORN) FROM 1915 TO 1921 INCLUSIVE, FOR URBAN (SOLID LINES) AND RURAL (DOTTED LINES) PARTS OF THE POPULATION OF THE ORIGINAL BIRTH REGISTRATION STATES

The four lower lines in the diagram give, for the same groups, the estimated percentage of women aged 15 to 44 inclusive.

for the total original birth registration area states are given in table 68. These figures being comparable throughout we may proceed to examine their trends.

In figure 39 are plotted, on an arithlog scale so that slopes may be visually compared, the course of indices *A* and *B* (native-born and foreign-born) for urban and rural areas.

From these data we note:

1. The general slope of the indices for the foreign-born over the whole period covered is slightly but consistently *downward*. That of the indices for the native-born is definitely *upward*. In all cases the 1918 influenza epidemic made, of course, a jog in the line, but this may fairly be disregarded in estimating the general slope or trend. So doing, we see that both foreign-born indices were *higher* in 1915 than in either 1920 or 1921, while both of the native-born indices were *lower* in 1915 than in 1920 or 1921. In other words, during the period under review the foreign-born index tended to decrease, while the native-born index tended to increase.

2. The native-born rural population maintained throughout the period a higher vital index than the native-born urban population. This relation is just reversed in the foreign-born, where the urban population has the higher index consistently. These findings confirm a conclusion tentatively reached in the earlier study.

Before any final conclusions can be drawn, however, as to the meaning of the trends noted, it will be necessary to examine into the question of the changes during the same period of time, if any, in the proportion of women 15 to 44 years of age in the population. To this end we have calculated for the period 1915 to 1920, by interpolation from the data of the 1910 and 1920 censuses, for the original birth registration states, the percentages of native-born and foreign-born women in urban and rural areas and plotted these lines in figure 39. It was assumed in this computation that the change from 1910 to 1920 in this part of the age composition of the population was linear. This is probably not absolutely true, but it is the best approximation that can be made from available data, and probably indicates the *general* trend of events with sufficient accuracy.

From these lower lines it is seen that the percentages of women 15 to 44 years of age in the four classes have changed but very slightly in the period 1915 to 1921. The changes are by no means great enough in amount to explain the trends in the vital indices shown at the top of the diagram. Furthermore, such slight trend as there has been in the proportion of women in the child-bearing ages has been uniformly *downward* in all four groups. Thus it is evident that if the changes in this proportion in the period were great enough in magnitude to explain the downward trend of the indices for foreign-born, which they are not, we should still be confronted by the fact that the increase in the indices for native-born has gone on in spite of a progressive tendency towards a more unfavorable age distribution of women in this class of the population.

The conclusion to which one must come is that the trends of the vital indices for the native and foreign-born moieties of the population do not find their explanation in any concomitant alteration in the proportionate number of women of child-bearing age in the population.

We may next examine the facts regarding the New England states, where the native-born populations, with the exception of Vermont, were

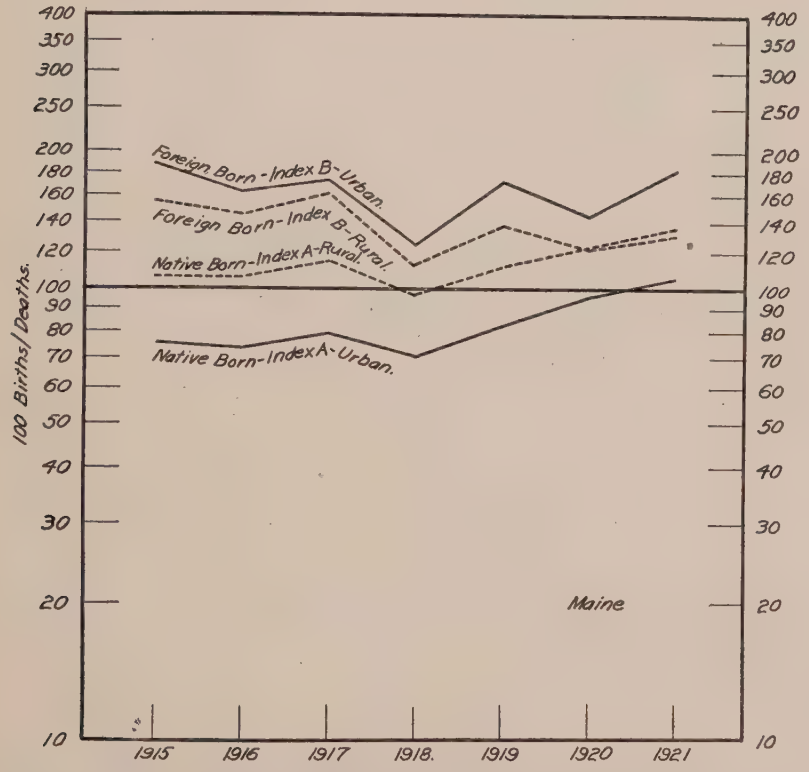

FIG. 40. THE COURSE OF THE VITAL INDICES *A* AND *B* (NATIVE-BORN AND FOREIGN-BORN) FROM 1915 TO 1921, INCLUSIVE, IN MAINE

Significance of lines as in figure 1

either not reproducing themselves or only barely so in the years covered in the earlier paper. The vital indices for the native and foreign-born, urban and rural parts of the population of Maine are shown in graphic form in figure 40.

It is evident that the same trends which were observable in the original registration states as a whole also hold in Maine. The urban native-

born line shows an upward trend sufficient to bring it above 100 in 1921, a condition not before realized since 1915. Furthermore the movement of the two rural lines has been such that the foreign-born index fell *below* the native in 1920 and that relative position was maintained in 1921. Altogether the situation as regards the biological condition of the population of Maine was much more hopeful in 1921 than it was in 1915.

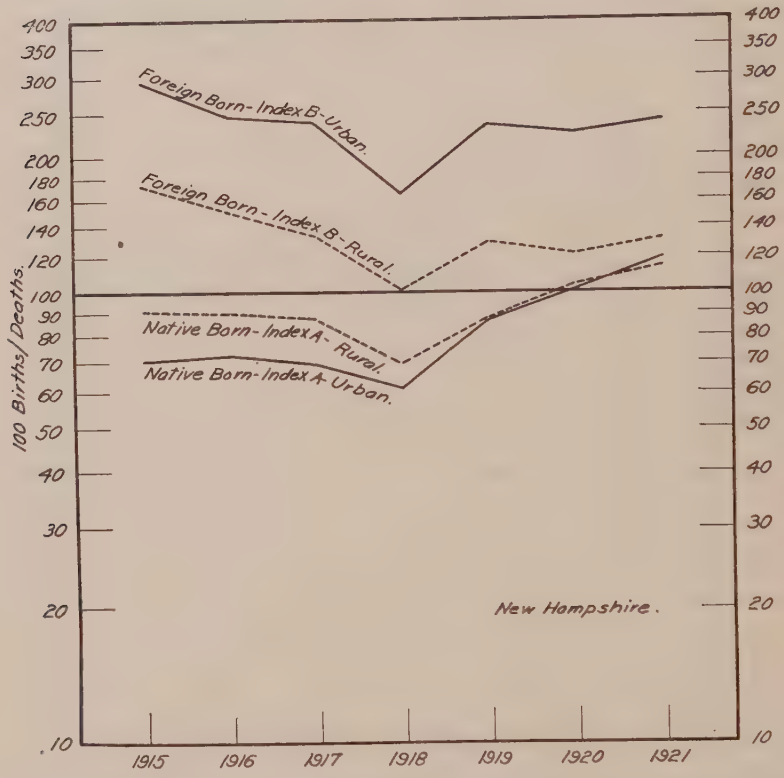

FIG. 41. THE COURSE OF THE VITAL INDICES *A* AND *B* (NATIVE-BORN AND FOREIGN-BORN) FROM 1915 TO 1921, INCLUSIVE, IN NEW HAMPSHIRE

Figure 41 presents the data for New Hampshire.

Here again the same general state of affairs is exhibited. The vital indices of the foreign-born population generally dropped from 1915 to 1921, and the indices for native-born rose. During the whole period up to 1920 both native-born indices had had values below 100. In 1920 they crossed the 100 line, and went still higher in 1921. The index for the

foreign-born rural population was in 1921 down close to the values for the native born parts of the population. This foreign-born rural line will cross the native-born lines in the near future if present tendencies continue. The foreign-born urban population has throughout a high vital index, but in the whole period, 1915 to 1921, its trend is clearly downward.

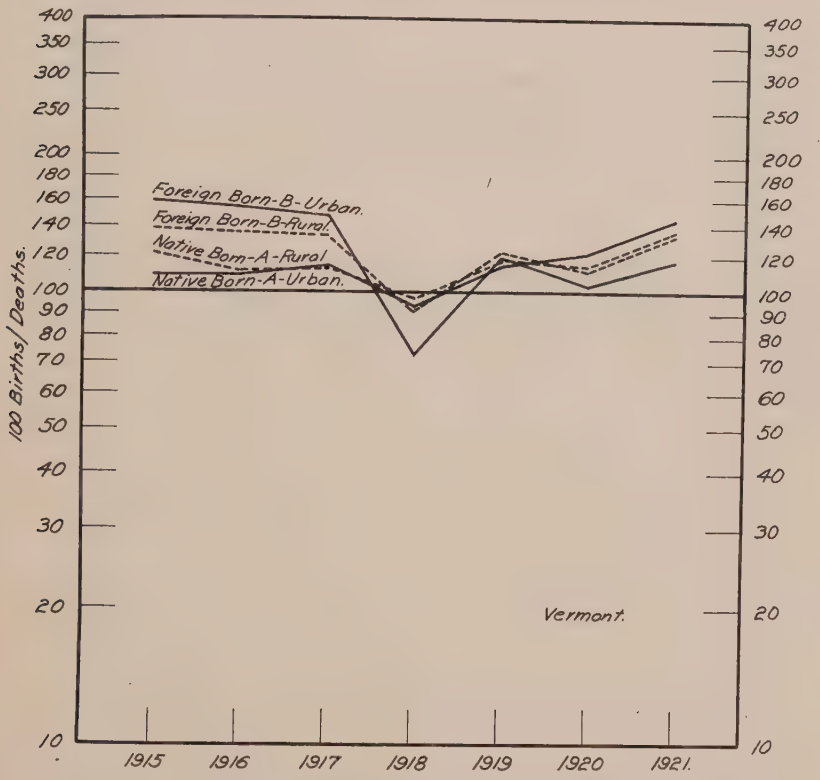

FIG. 42. THE COURSE OF THE VITAL INDICES *A* AND *B* (NATIVE-BORN AND FOREIGN-BORN) FROM 1915 TO 1921 IN VERMONT

The data for Vermont are given in figure 42.

Here all four groups have vital indices which are obviously not widely divergent in their values. The native-born urban group shows an obvious upward trend in the period, and the foreign-born urban group a downward trend. The two rural indices exhibit little change in the period, but what there is follows the same course as in the urban groups. It is

of interest to note how much more severely the foreign-born urban group was affected by the influenza epidemic than was any other element in the population.

Figure 43 presents the data for Massachusetts.

Here the usual relation between the two native-born groups is reversed.

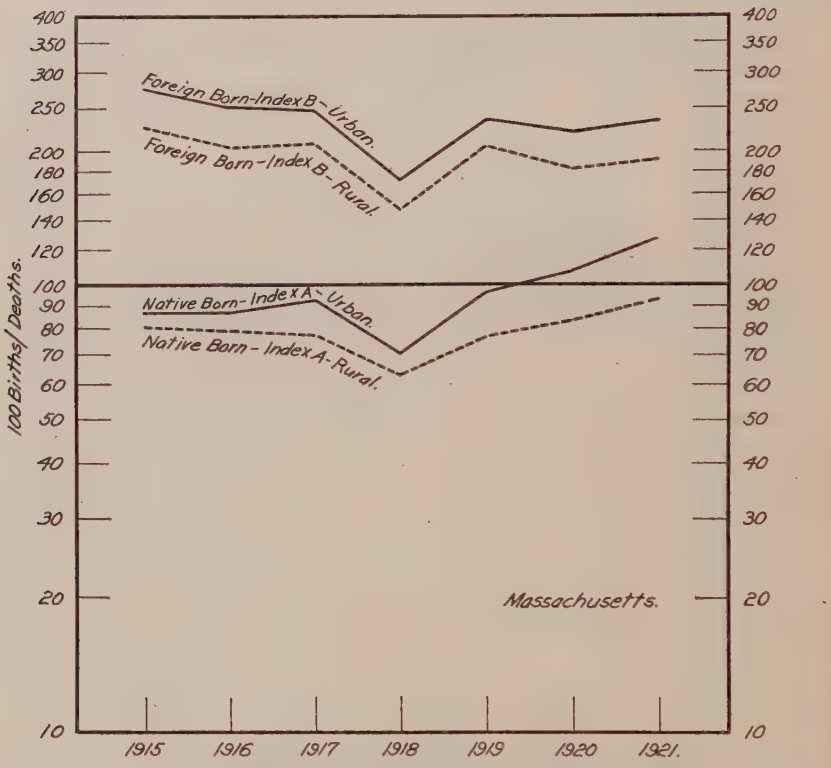

FIG. 43. THE COURSE OF THE VITAL INDICES *A* AND *B* (NATIVE-BORN AND FOREIGN-BORN) FROM 1915 TO 1921 IN MASSACHUSETTS

The rural native population has a lower vital index throughout than the urban. The trends are of the same sort as in the other cases but not marked in degree. The native-born rural line lies entirely below the 100 line, although in 1921 the index was above 90. Altogether it may fairly be said that the favorable tendencies which have been noted in the other New England states and in the group as a whole, are less striking in Massachusetts.

Figure 44 gives a picture of the Connecticut population which in all essentials is identical with that of Massachusetts.

Summing up the whole case to this point it may be said that there seems to be clear evidence that the vital index has tended to increase for the native-born population and to decrease for the foreign-born population, in the period from 1915 to 1921 inclusive. This is true both for the original

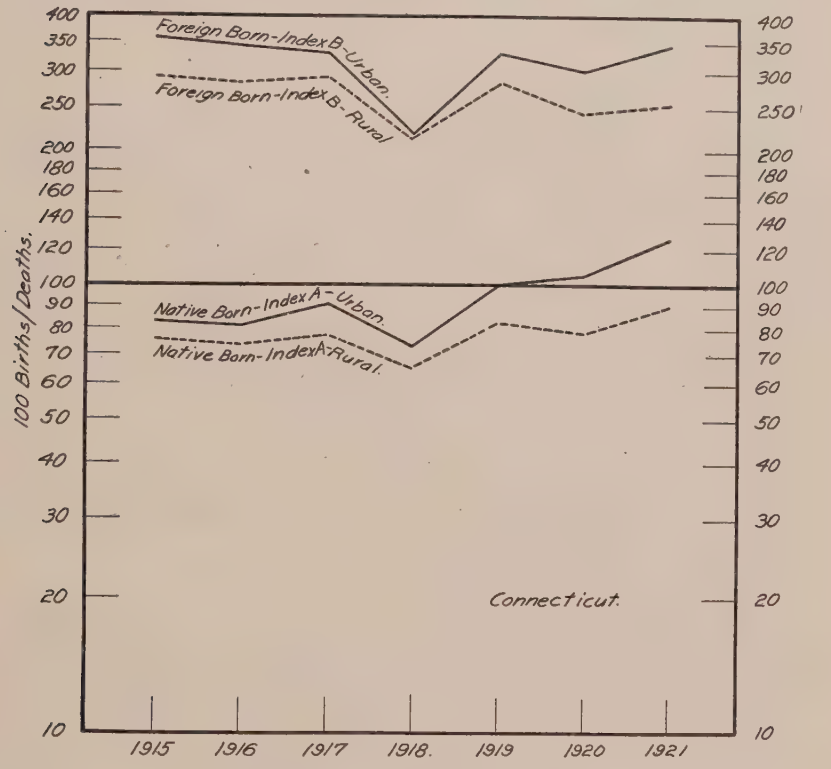

FIG. 44. THE COURSE OF THE VITAL INDICES *A* AND *B* (NATIVE-BORN AND FOREIGN-BORN) FROM 1915 TO 1921 IN CONNECTICUT

birth registration states as a whole group, and for each of the New England states in which complete records exist, when taken separately. In this statement due allowance is made for the large dip in the curves due to the influenza epidemic of 1918. These trends cannot be explained as due to any change in the proportion in the population of women in the child-bearing ages. Seven years is obviously too short a period to justify

any dogmatic or sweeping conclusions as to trends. But it *suggests*, for future verification, that the native-born population in the northern and eastern sections of the country is biologically improving, while the foreign-born population is becoming less sturdy biologically. So far as they go, and can be regarded as reliable, these are hopeful tendencies.

Biologically the major cause of these trends is a more rapid decline of death rates than of birth rates in the period. This should be an encouraging result to health officials and others interested in the reduction of the rate of mortality.

Let us now consider the second large problem presented by our vital indices. This is the question of the trend of the vital index in the negro population. Here we encounter at the outstart a material difficulty. The District of Columbia is the only demographic unit in table 67 for which there is a continuous record from 1915 to 1921, and in which there is a large negro population. Here the population is entirely urban. The best that can be done in the way of any general approach to the negro vital index problem is to set up a group of southern birth registration states, realizing fully that the composition of this group changes in the different years, and being correspondingly cautious in drawing conclusions. Such a southern birth registration states group includes the District of Columbia, Kentucky, Maryland, Mississippi, North Carolina, South Carolina, and Virginia. Of these states two only, Mississippi and South Carolina, can be regarded as falling within the so-called "black belt." The others are border states.

The vital indices for this group of states are given in table 68.

In figure 45 is given a comparison of the negro and white indices for this southern group. The indices for rural populations necessarily begin only with 1916. On the diagram are also plotted the percentages of women 15 to 44 in the total female population of similar kind, for the four different groups.

This diagram clearly indicates an upward trend of all four vital index lines. In the rural portions of the population the slopes upward are marked in degree. They are less so in the urban portions. The index for rural whites in these southern states was nearly twice as large in 1921 as in 1916. Essentially the same was true of the rural negroes, though at all times their index was well below that of the corresponding class of whites. The index for the urban negro population has a distinct upward trend over the whole period, and became in 1920 slightly greater than 100, and in 1921 quite definitely so.

These marked upward trends in the vital indices in these southern states cannot be accounted for by the small changes which have occurred in the period in the proportion of women in the population of the ages 15 to 44. Such slight change as has occurred in this proportion has been

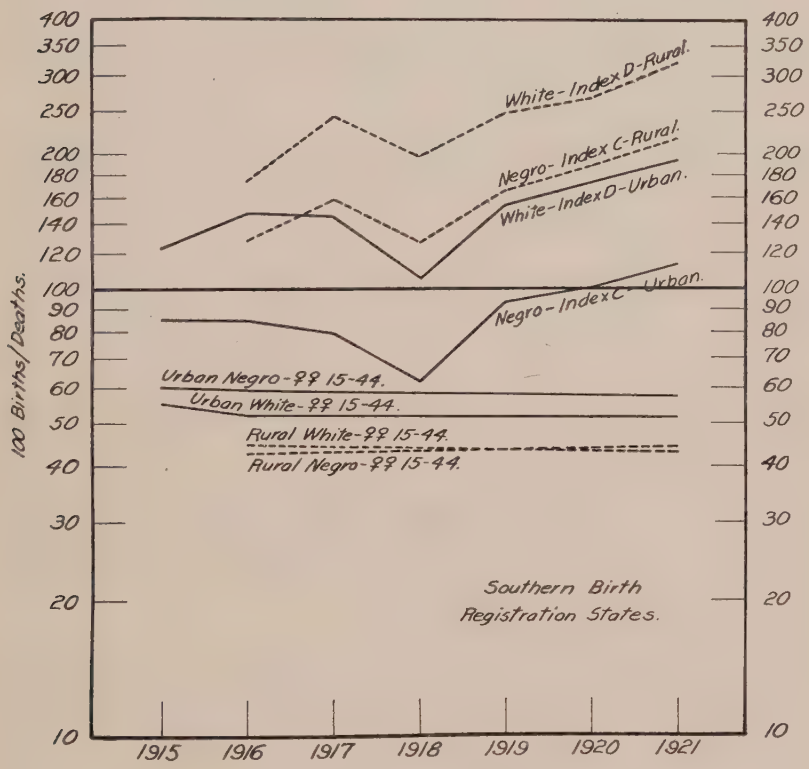

FIG. 45. THE COURSE OF THE VITAL INDICES *C* AND *D* (NEGRO AND WHITE) FROM 1915 TO 1921 INCLUSIVE FOR THE URBAN (SOLID LINES) AND FROM 1916 TO 1921 INCLUSIVE FOR THE RURAL (DOTTED LINES) PARTS OF THE POPULATION OF A GROUP OF SOUTHERN AND BORDER STATES

The four lower lines give, for the same groups, the estimated percentage of women aged 15 to 44 inclusive.

in a direction opposite to that requisite to explain a rising vital index, in all but one case, the rural negro population. But there the upward shift of the proportion of women in the age groups from 15 to 44 has been only between 1 and 2 per cent, an amount obviously too small to explain the great increase in the vital index.

A purely statistical factor which does play some part in causing the upward trend of the index lines in figure 45 is improving registration of births in this area. How important a factor this is it is difficult to estimate. It is, however, interesting to note that, on the whole, the negro and white lines in figure 45 are about parallel. From this fact one may tentatively infer that probably the accuracy of birth registration is being increased at about the same rate in both negro and white moieties of the population of these states.

An examination of the birth and death rates separately for the states in this southern group indicates clearly that the main factors in the upward trend of the vital index is declining mortality, and not increasing birth rates. The *recorded* birth rates are in general increasing in these states, probably in chief part as a result of improving registration. But these increases are on the whole distinctly smaller in amount than are the decreases in the death rates in the same states. The International Health Board has been very actively working during the years covered in this study to improve general health conditions in the south. That their efforts in this direction have greatly stimulated local health authorities cannot be doubted. However the credit is to be distributed, the fact is certain that the death rates have fallen markedly in these states in the last few years, in the negro as well as the white population. It is apparent that a continued improvement of the health conditions and the mortality of the negro may give an aspect to the race problem in this country, entirely different to that which it has hitherto borne.

That it is the falling death rate rather than an increasing birth rate that is chiefly responsible for the upward trend of the index is confirmed if we examine the homogeneous urban population of the District of Columbia over the whole period.

The facts are displayed graphically in figure 46.

The same upward trend is shown as in the case of the whole group of states. It cannot be accounted for by changing age composition of the population. During the period the birth rate in either white or colored did not change significantly in either direction. But the white death rate fell from 15.1 in 1915 to 12.1 in 1921, and the colored from 26.2 in 1915 to 18.9 in 1921.

Summarizing the case it may be said that the native white populations seem generally to be in a period of biological improvement, judged by the vital index. The foreign-born whites are, on the contrary, displaying progressively less biological vigor by the same criterion. In the south the vital index is increasing in value year by year, both in white and col-

ored, chiefly as a result of generally decreasing mortality rates, but partly as a result of improving birth registration. The vital index of the negro population is not gaining on that of the white. Both maintain their relative positions. The urban negroes have a very low vital index; the rural negroes one slightly higher than that of the urban whites, but much below that of the rural whites. The underlying reason for the upward trend of the indices for native white and colored populations is chiefly to be found in the fall in the death rates which has occurred during the

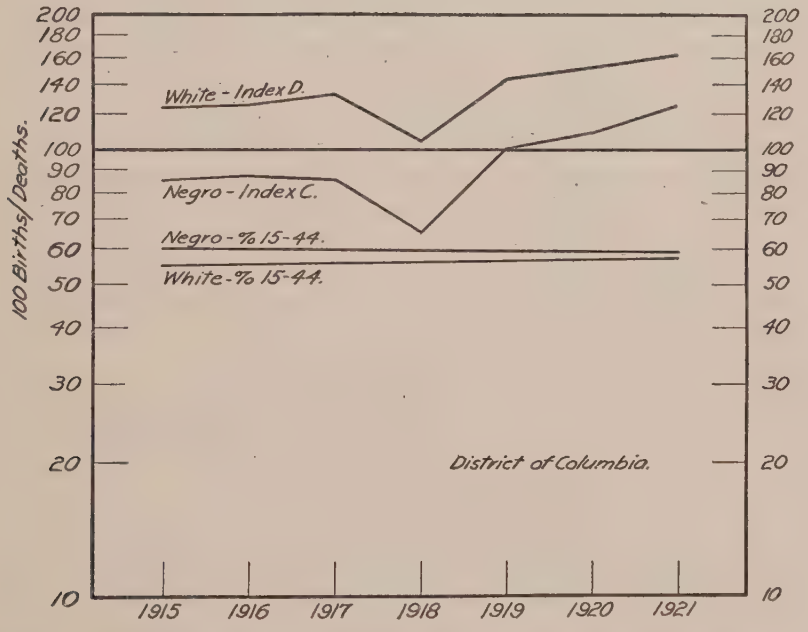

FIG. 46. THE COURSE OF THE VITAL INDICES *C* AND *D* (NEGRO AND WHITE) FROM 1915 TO 1921 FOR THE POPULATION OF THE DISTRICT OF COLUMBIA, TOGETHER WITH THE ESTIMATED PERCENTAGES OF WOMEN 15 TO 44

period, and with quite unprecedented rapidity since 1919. It seems unlikely that the trends displayed during these seven years in the vital indices will continue indefinitely, for two reasons. In the first place it would be rash to hope that the decline in the general death rates can go on at the rapid rate which characterized the period from 1919 to 1921. There is no reason to suppose that it may not continue to decline, at least for a long time, but there is much reason for supposing that its rate of decline will be retarded. In any case, constantly improving conditions

of public health and their reflection in the mortality returns, have taken away any force which might at some time possibly have been thought to attach to the "race suicide" argument against the falling general birth rate. Any population or group that year by year is producing more than one baby for each death is not on the high road to extinction, whatever other deficiencies it may have.

Differential fertility, and still more differential survivorship in the evolutionary sense not the actuarial, is the most disturbing thing. The populations in the relatively highly industrialized parts of the country exhibit vital indices for the foreign-born elements rather distressingly out of line with those of the native elements. But it seems to me easily possible to be too pessimistic about this situation. It is reasonable to believe that never again will wholly unrestricted immigration into this country be permitted. On the contrary it is probable that restrictions will be unremittingly more drastic and extensive. Doubtless these measures whatever they are will be thought by some persons to be unjust and unscientific, and doubtless they will be, in some measure at least. But probably they will *restrict*, and that appears to this particular biologist the really important point in the case. I have still very great confidence in the absorptive and assimilative powers of the American people for queer non-American bipeds provided they do not come to us too fast. This confidence let me hasten to state is based almost wholly upon our size and not upon our wisdom or judgment in dealing with the problems of society. If for a period, even so short as ten years, we had the same kind of restriction of immigration for all races as we now practice for the Chinese, it is probable that at the end of the time there would be very little worrying about the foreign-born population. No politician would dare advocate this policy now. We shall approach it, slowly, inefficiently, but very certainly. What it really means in terms of social values is that the time is now hard upon us when the counsel of enlightened self interest will be to make America a land of opportunity for Americans, rather than for the world at large.

CHAPTER X

Constitution and Tuberculosis[1]

THE PROBLEM

The problem to which attention is directed in this chapter is that of the etiology of clinically active tuberculosis. A few deny the existence of any such problem. Happy in that superlative self-esteem vouchsafed in full measure only to ardent worshippers at the shrine of *idola fori*, they would, if they could, stop all true research on the problems of tuberculosis, and give countenance only to such so-called investigative activities as are guaranteed beforehand not to run counter in any particular to the plentiful exhibition of their special brand of eye-water, blatantly touted as the only true panacea for the ills tuberculosis engenders. Fortunately, all scientific students of tuberculosis realize keenly that the problem of the etiology of the disease is a real and important one. The case cannot be better put than it is in a recent paper by Lawrason Brown and his associates[2] where they say, "Our hope in publishing these experiments is that others may realize that the etiology of tuberculosis is not a closed book, but one that contains many disconcerting and confused pages that need to be rewritten."

The problem may with some precision be defined by a statement of the following well-known and thoroughly established facts:

1. Very few, if any, persons escape infection with tubercle bacilli at some time in their lives, especially if they live in large industrial cities. Fishberg,[3] after reviewing the literature on the subject says, "No matter what the cause of death may have been, whether the persons knew that they had been tuberculous or not, between 50 and 100 per cent of people living

[1] The material in this chapter first appeared under the title, "The relative influence of the constitutional factor in the etiology of tuberculosis," in the *Amer. Rev. Tuberc.*, vol. 4, pp. 688–712, 1920.

The paper was read before the North Atlantic Tuberculosis Conference at Richmond, Va., October 7, 1920, in a symposium on the general topic, "The basic cause of breakdown with tuberculosis."

[2] Brown, L., Petroff, S. A., and Pesquera, G., Etiological studies in tuberculosis, *Amer. Rev. Tuberc.*, 1919, iii, 621.

[3] Fishberg, M., *Pulmonary Tuberculosis*, Lea & Febiger, Philadelphia and New York, 2nd edition, 1919, p. 58.

in large cities show active, quiescent or healed tuberculous lesions in some organs of their bodies."

2. A much smaller number of persons than are known and can be proved to have been at some time or other infected with tubercle bacilli ever develop clinically recognizable tuberculosis.

3. Of the two moieties of the infected, those on the one hand who do, and those on the other hand who do not develop the disease in clinically active form, many can be readily shown to have lived under essentially or statistically the same environmental circumstances.

What factors determine the group into which a *particular* infected

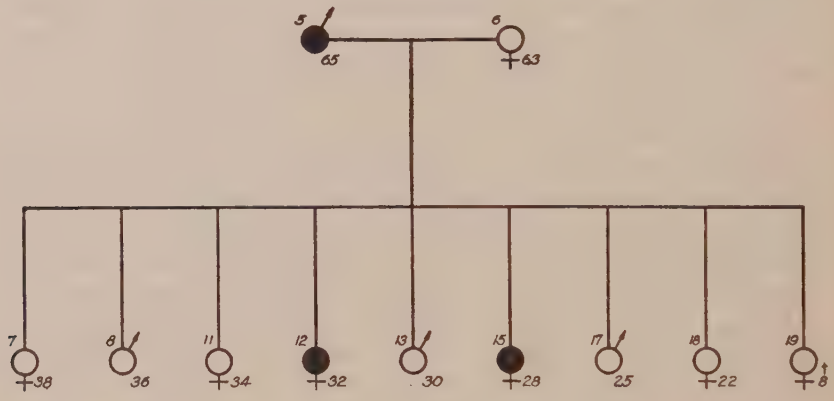

FIG. 47. PEDIGREE CHART OF A TUBERCULOUS FAMILY

Where the body of the sex sign is solid the person has tuberculosis of the lungs; where it is open the person is not tuberculous. The numbers to the right of the bottom of each sex sign indicate the present age, or age at death of the person indicated. The numbers to the left of the top of the sex signs are the designating numbers (in lieu of names) of the indicated persons. † denotes that the person is dead.

person shall fall, and what is the quantitative influence of each particular possible factor in making this determination?

This I conceive to be the fundamental and essential problem of the etiology of clinically active tuberculosis.

The problem can be still more precisely visualized by the consideration of a particular family taken from one of the pedigrees worked up in my laboratory.

The time relations of contact of the children in the tuberculous family, whose pedigree is given in figure 47, with their tuberculous father, and with each other, are given in summary form in table 69.

The essential facts regarding this family, as of 1919, the time of their investigation, are as follows: The father, individual 5, is a laboring man who has always been addicted to the use of alcohol in considerable quantities. In 1886, when he was thirty-two years old, he developed clinically active pulmonary tuberculosis and had frequent hemorrhages. He was in this condition when child 12 was born. The firstborn daughter, no. 7, has always been at home with the family down to the present time, having thus lived thirty-three years in close contact with an active case. She is by occupation a seamstress. She has never developed any indications of tuberculosis. The first son, individual 8,

TABLE 69

Contact relations involving individuals in pedigree chart

INDIVIDUAL NUMBER	BORN	BECAME TUBERCULOUS	YEARS LIVED IN CLOSE CONTACT IN SAME HOUSEHOLD WITH CASES BEFORE OR WITHOUT ACQUIRING TUBERCULOSIS
5	1854	1886	
6	1856		33
7	1881		33
8	1883		25
11	1885		20
12	1887	1913	26
13	1889		25
16	1891	1912	21
17	1894		24
18	1897		22
19	1880		8

was born in 1883 and lived at home in the same household with his father until he was twenty-eight years old (twenty-five years familial contact). He has never developed any symptoms of tuberculosis. The next child, no. 11, was born in 1885 and lived at home with the family, in daily close contact with her tuberculous father for twenty years. She has never developed any symptoms of tuberculosis. The next child was born in 1887. In 1913, when she was twenty-six years old, she developed clinically active pulmonary tuberculosis after having lived twenty-six years in the same household and in close contact with her tuberculous father. Individual 13 was born in 1889 and lived for the first twenty-five years of his life in the same household with the tuberculous father. He has never shown any symptoms of tuberculosis. Individual 16 was born in 1891, and in 1912, when she was twenty-one years old, developed clinically

active pulmonary tuberculosis. She had lived all this time, and still does, in the same family with her father. Individual 17 was born in 1894 and has always lived with the family until he was drafted and went into the American Expeditionary Force and to France. Since his discharge he is again living with the family. He has never developed any symptoms of tuberculosis, although he has lived for roughly twenty-four years of his life in close daily contact with his father, and also with his two sisters, since the time when they became tuberculous. Individual 18 was born in 1897 and has always lived in the same household with her father and the rest of the family. She is now twenty-two years old and thus has had close intimate household contact for twenty-two years with active tuberculous patients. Individual 19 was born in 1880, and died when eight years old, probably of acute nephritis, but certainly not of tuberculosis. Individual 6, consort of the tuberculous father, has lived in close and intimate household contact with him for the thirty-three years since he first gave evidence of clinically active tuberculosis. She has never shown any symptoms of any tuberculous disease.

Putting all the facts in regard to this family together what do we see? Out of eight living children, all of whom lived together in the same environment, and had the same food, for twenty or more years, two have developed clinically active tuberculosis, while the other six have shown no signs whatever of the disease. All have been in close and intimate personal family contact with their father, who has, as our careful investigation of the family life and medical history indicates, been an open tuberculous patient during the major portion of that time, unable to do any work for most of the time, with hemorrhages recurring at irregular but fairly frequent intervals. Furthermore, individuals 12, 16, and 18 have for many years slept together in the same room. Numbers 12 and 16 have been tuberculous for six and seven years respectively, while no. 18 has never shown any symptoms of the disease.

It is to be presumed that every member of this family was long ago infected with tubercle bacilli. If we attribute breakdown to poor diet, or to bad housing conditions, it is difficult to see why the disease should have flared up in clinically active form in only two out of eight children who have lived in this same environment throughout the period. All have been subjected to substantially the same degree of environmental and economic pressure. They have all had to work hard, because the father has contributed very little to the support of the family in all these years.

Such a case as this, which could be duplicated over and over from our family records, throws into sharp relief what I conceive to be the essential

problem of the etiology of tuberculosis. Out of an equally infected group of individuals, equally in close contact with active and open tuberculosis, and living under identical environmental conditions, some individuals in the group develop clinically active tuberculosis while others never do. What is the reason for the differentiation? This is the kernel, as it seems to me, of the problem.

The investigation on which this family pedigree which we have been discussing is based was made in 1919. It may be objected that all of the children, nos. 7, 8, 11, 13, 17, and 18, will presently develop tuberculosis and die of it. No one can assert, of course, that this will not happen because it is an event which belongs in the future. I conceive it to be improbable, however, that such an event will occur. The force of untoward environmental pressure on these individuals during the past twenty years has been about as great as it could be, and certainly greater than it is likely to be in the future.

Another objection which might be conceivably raised to the illustration is that neither individual 5, 12, nor 16 has tuberculosis. The answer to this is that they have repeatedly had diagnosis and treatment of the disease in sanatoria and dispensaries. If they are not victims of the disease no one is.

I wish it to be quite clearly understood that this pedigree is introduced here only as an illustration of the problem, and not in any sense, taken by itself, as evidence of the influence of the hereditary factor in the etiology of the disease. It obviously suggests such a factor, but suggestion and demonstration are usually a long way apart. It does illustrate with great clearness and precision the nature of the problem facing one who would account for the etiology of clinically active tuberculosis.

HOW SHALL THE PROBLEM BE ATTACKED?

It is obvious on purely *a priori* grounds that in a broad sense the cause of the outbreak of clinically active tuberculosis in one moiety of a group of infected individuals and not in the other moiety must fall into one of the three following categories:

1. Environmental. There are significant differences in the environmental forces, using the term in the broadest sense to include biological as well as physical environment, impinging upon the two moieties, these differences being of sufficient magnitude alone to determine the observed difference in susceptibility to tuberculosis.

2. Constitutional. There are significant differences, of a germinal, hereditary character, in the innate biological constitutions of the indi-

viduals composing the two groups, and these differences alone determine the differentiation in respect of outbreak of active tuberculosis.

3. Both. The two groups differ in both environmental and constitutional respects, neither factor alone being ordinarily determinative of tuberculous breakdown.

The first of the enumerated views is the popular one. It opens large possibilities for altruistic enterprise. I judge from what I hear and read that there are still extant naïve individuals who quite honestly believe that if everyone could be induced and enabled to drink two quarts of milk a day and sleep with his windows open, tuberculosis—certainly pulmonary tuberculosis—would disappear from the face of the earth by the middle of next June at the very latest. If only these good souls were right, how happy we all should be!

The second view is extremely unpopular. Any doctrine which implies that the constitutional weaknesses, whether patent or hidden, of the father might be visited upon the sons stirs up no furious enthusiasm in this day and age. The person who has, or had, a number of tuberculous near relatives finds himself particularly antipathetic to this view. If indulged in, what becomes of one's hope in the two quarts of milk or the open window?

The relative popularity of a viewpoint happily has nothing to do with its validity. Extremists in any direction are usually wrong. It is altogether improbable that either environment or hereditary constitution *alone* determines the differential incidence of active tuberculosis. It is much more likely that always both factors are involved. The task is, as I conceive it, not to answer the question "Is it environment?" or "Is it heredity?" but rather to measure, with the greatest attainable precision the relative influence of each factor in determining the result.

In earlier days constitution was generally regarded by medical men as playing a very important rôle in the etiology of tuberculosis. The most comprehensive and critical discussion of the older literature on this point has been furnished by Bulloch and Greenwood.[4]

There are only a few chief lines yet thought of by which one can get quantitative evidence as to the relative importance of the hereditary factor in the etiology of tuberculosis. We may direct our attention briefly to some of these different lines of evidence, it being understood, of course, that in the limitations of space which are imposed upon this chapter, it will be impossible to deal with the matter in more than the most sketchy

[4] Bulloch, W., and Greenwood, M., The problem of tuberculosis considered from the standpoint of disposition, *Proc. Roy. Soc. Med.*, May, 1911, iv, Epidem. Sec., 147–184.

fashion. In other words, what we shall try to do is to present something about the *kinds* of evidence, rather than the amount of each kind which may be brought to bear in any consideration of the problem.

1. Direct evidence as to the inheritance of the tuberculous diathesis. Here the most important studies up to the present time which are deserving of serious scientific consideration are those made in Pearson's[5] laboratory, or under his direction. Pearson has attacked the problem of the relative influence of heredity and environment in the etiology of tuberculosis from a number of different angles. In this section we shall consider only one, namely, that side of his work in which he attempts to measure directly the degree of inheritance of the tuberculous diathesis from parent to offspring. This is done by the method of correlation. The coefficient of

TABLE 70

Coefficients of correlation between parent and offspring

CHARACTER		CORRELATION	AUTHORITY	DATA
Physical	Stature	0.51	Pearson and Lee	Pearson's F. R.
	Span	0.46	Pearson and Lee	Pearson's F. R.
	Forearm	0.42	Pearson and Lee	Pearson's F. R.
	Eye color	0.50	Pearson	Galton's F. R.
Pathological	Deaf-mutism	0.54	Schuster	Dr. Fay's
	Insanity	0.53	Heron	Dr. Urquhart's
	Insanity	0.47	Goring	Convict prisons
	Phthisis	0.50	Pearson	Dr. Rivers's
	Phthisis	0.50	Goring	Convict prisons

correlation between parent and offspring in respect of presence or absence of pulmonary tuberculosis is determined by well-known methods, and it is assumed that the correlation which is found is due to inheritance. Unfortunately, the series dealt with were not as large as would be desirable from a statistical standpoint. The general results attained by Pearson for the correlation between parent and offspring in respect to existence of pulmonary tuberculosis are shown in table 70.

From this table it will be seen that, on the face of the facts, the indication is that the tuberculous diathesis is inherited in about the same degree from parent to offspring as such physical characters as stature, forearm length, eye-color, etc. The contention of the environmentalists and

[5] Pearson, K., Tuberculosis, heredity and environment, *Eugenics Laboratory Lecture Series*, 1912, viii. This is a popular summary of the work mentioned.

infectionists, however, upon the appearance of these results, was at once that such correlations might well arise solely by familial infection, rather than true inheritance.

Pearson has attempted in a number of ingenious ways to meet this point. Whether one considers that he has successfully done so or not depends, one gathers from the literature, in part upon the nature and degree of one's prior prejudices in the matter. It will, however, pay us to look at a few of the facts which Pearson has brought forth even though we do not accept them at their face value. If the explanation of the observed correlation between parent and offspring in respect of tuberculosis is familial infection rather than similarity of biological constitution, it has been contended by Pearson that the correlation between husband and wife in respect of tuberculosis ought to be of something approaching the same order of magnitude as that shown for parent and offspring. If the reason why a tuberculous man's children have tuberculosis in greater proportion than the disease occurs in the general population, is because he has infected them as a result of close familial contact, by the same token it is held by Pearson that his wife ought to be tuberculous. The only quantitative difference, which might possibly be expected, would be such as arose from the fact that when a consort came in contact with the familial tuberculous infection, he or she would be at a more advanced age. It might be contended that at such age the individual would perhaps be better able to resist such infection than the children born in the family and subjected to this immediate and massive source of infection from the earliest ages. Leaving aside any attempt at explanation, the fact is that the resemblance between husband and wife in respect of tuberculosis is as follows (quoting from Pearson):

		Coefficient of correlation
Husband and Wife	All poor	−0.01
Husband and Wife	Prosperous poor	+0.16
Husband and Wife (Pearson)	Middle classes	+0.24
Husband and Wife (Dr. Williams)	Professional classes	+0.28

It would thus appear, either that like mates with like more commonly in the more intellectual classes, or that infection is more likely to occur in middle class than in poor households. I think there is not the least doubt that much of the relatively small resemblance of husband to wife in the matter of phthisis is due to a selective influence and not to infection at all. This selection is largely an intellectual one and has no existence among the very poor.

Pearson went on to show from his family records, leaving entirely out of account the husbands and wives themselves, and determining the degree of assortative mating between tuberculous *stocks*, that the correlation

worked out to 0.30, as high as in the extreme cases of mating with regard to pulmonary tuberculosis.

Now if we compare the correlation coefficients for parent and offspring as shown in table 70, with those for husband and wife just given, it would indicate that the parent is twice as dangerous to the offspring, if the source of the resemblance is familial infection, as the husband is to the wife. This result is held by Pearson to indicate that neither correlation is primarily to be explained on the basis of infection, but that the one is due to inheritance of the diathesis, and the other to assortative mating of tuberculous stocks.

The whole case, however, leaves one with the feeling that so far as the direct measurement of inheritance is concerned, we need more and better evidence than that which has been furnished by Pearson. We need, in the first place, more critically accurate original data, in which we shall have exact and direct information, in so far as it is possible to get it, obtained by trained field workers in the families, regarding such points as degree and duration of familial contact with open cases, as well as the more usual points upon which data are taken. At the same time, we need equally critical data from nontuberculous families collected in the same way by field workers in the families. Again it is not entirely clear from a methodological viewpoint that the correlation method is a sound one, taken by itself alone, for the measurement of the intensity of inheritance. The results got in this way need confirmation by some independent method of analysis before they can be unreservedly accepted at their face value. Pearson's was pioneer work, and it unquestionably suggests that the hereditary factor is a highly important one in this disease. Only the reckless environmentalist fanatics will be disposed to neglect it, or to regard it as wholly worthless. The problem to be solved is, however, one of such vast importance to the human race, that before any sweeping conclusions are drawn on which administrative action is likely to be based, it is essential that much further investigation of the problem be made. Furthermore, any statistical or family history evidence on the subject should be checked and verified by direct experimental evidence in the laboratory, obtained from breeding operations with lower animals, as to the inheritance factor in the etiology of tuberculosis. It is a great satisfaction to know that investigations of this kind are now in progress.[6]

[6] *Cf.* Wright, S., and Lewis, P. A., Factors in the resistance of guinea-pigs to tuberculosis, with especial regard to inbreeding and heredity. *Amer. Nat.*, vol. 55, pp. 20–50, 1921.

In order to illustrate directly some of the difficulties which inhere in any attempt to make a thorough scientific investigation of the problem of the inheritance of the tuberculous diathesis in man, I should like to speak briefly about some of the work going on in my own laboratory. About five years ago plans for a comprehensive and searching investigation of the problem of the etiology of clinically active tuberculosis were embarked upon.[7] While it was contemplated that the problem would be approached from many angles, and this has in fact been done, I wish to speak now of one phase of the work, namely, that which has to do with the obtaining and analyzing of data directly bearing upon the problem of inheritance of the diathesis. The plan of the work was to make a thorough study of the family histories of individuals afflicted with tuberculosis, and of individuals free from tuberculosis, both sets of individuals being taken from the same economic and social strata. "Family history" in this connection is used in the broadest possible sense to include not only information as to relatives direct and collateral, but also as many pertinent data as possible about the individual life histories and habits of these individuals.

The work is in far too early a stage even now to make any statement of results whatever, nor shall I here go into any discussion of the details about either the comprehensiveness or the thoroughness of the records which have been accumulated (*cf.* on this point Chapter XII *infra*). I may merely say in passing that, so far as I am acquainted with the facts, I know of no data for the study of the etiology of this or any other disease which begin to approach these in respect of either comprehensiveness, detail, or accuracy. Our ideals in these respects were set very high, and in consequence a number of years must still elapse before the material can be completely analyzed. Here I wish to use a little of the material, only for the purpose of illustrating the difficulties which beset research upon this problem.

If heredity is a factor of importance in the etiology of clinically active tuberculosis it would be reasonable to expect that a tuberculous individual would have a larger proportion of his or her blood relatives tuberculous, both direct and collateral and in ascending and descending generations from the individual, than would a person who was not tuberculous. Or, to put the matter in another way, suppose we stopped the first man or woman we chance to meet on the street and ascertained by appropriate methods

[7] In the first year this investigation was supported by a grant from the National Tuberculosis Association. Thereafter it was maintained over an interim period by the Russell Sage Foundation, and finally brought to the conclusion of the record taking through a large grant from the Commonwealth Fund.

whether that person was or was not tuberculous, and at the same time made detailed inquiries as to his or her blood relatives. Should we be justified in laying a wager, if the individual proves to be tuberculous, that a larger percentage of his relatives will be also tuberculous than if he himself were nontuberculous, and what if any odds could we give in such a wager? In table 71 I have collected together the data on this point from 57 family

TABLE 71

Showing the frequency of occurrence of tuberculosis among the blood relatives of (a) tuberculous persons and (b) nontuberculous persons

(Figures based upon the family histories of 33 tuberculous and 19 nontuberculous subjects)

GENERATION AND GROUP RELATIVE TO SUBJECT OF HISTORY	NUMBER OF BLOOD RELATIVES WHO ARE			PER CENT TUBERCULOUS
	1. Tuberculous	2. Nontuberculous	Total	
Same generation:				
a. Tuberculous subject	96	876	972	9.9
b. Nontuberculous subject	5	979	984	0.5
Parental generation:				
a. Tuberculous subject	42	430	472	8.9
b. Nontuberculous subject	14	749	763	1.8
Grandparental generation:				
a. Tuberculous subject	7	236	243	2.9
b. Nontuberculous subject	8	298	306	2.6
Great grandparental generation:				
a. Tuberculous subject	3	30	33	9.1
b. Nontuberculous subject	1	86	87	1.1
Child generation:				
a. Tuberculous subject	43	972	1015	4.2
b. Nontuberculous subject	0	212	212	0.0
All generations:				
a. Tuberculous subject	191	2544	2735	7.0
b. Nontuberculous subject	28	2324	2352	1.2

histories, involving something over 5000 blood relatives of the 57 subjects of these family histories. Of these subjects, from each of which a detailed history starts, 38 are tuberculous, and 19 are nontuberculous. Each group may be regarded as a random sample of the working class population of Baltimore, the only differential factor in the selection being that in the one

case the individual with whom a history started was tuberculous, and in the other case not.

Five generations are included in the table. There are: First, the generation to which the subject of the inquiry himself belongs: this is the generation in which his blood relations are brothers, sisters, or cousins; second, the parental generation, that is, the generation to which the subject's father and mother, and his uncles and aunts belong; third, the grandparental generation; and fourth, the great grandparental generation. The "child generation" is the generation to which the subject's children, and his nephews and nieces belong. Relatives by marriage solely, of course, are not included. Only those persons are included who have a biological or "blood" kinship to the subject in some degree or other. But all *degrees* of kinship, collateral and direct, near and remote, are included.

The net result is striking. Taking all the generations together we see that in these statistics a tuberculous person has 7 per cent of his or her blood relatives tuberculous, whereas a nontuberculous person, chosen at random, has only 1.2 per cent of his or her blood relatives tuberculous, the absolute numbers involved in the two samples being approximately the same. In other words, in so far as this material may be regarded as typical, we can assert that a tuberculous person chosen at random from the working class population will have nearly six times as many blood relatives tuberculous as will a nontuberculous person taken at random from the same population. We note that the same kind of difference appears in each generation. The difference is insignificantly small in amount in the grandparental generation. This must be attributed, I think, merely to random sampling. I judge that with larger numbers, this generation would pass into the same class as the others.

One other point which needs explanation, lest an erroneous conclusion be inadvertently drawn from table 71, appears in the figures for the child generation. It will be noticed that whereas the histories of the 38 tuberculous subjects yield 1015 blood relatives in the child generation (F_1 generation in current genetic usage) the 19 histories of non-tuberculous subjects yield but 212 individuals in the same generation. This does *not* mean, as might be supposed, a reduced fertility in the nontuberculous as compared with the tuberculous. The difference arises merely from the fact that the method of selection of subjects upon which to start histories is such as to give a considerably lower average age of *subject* in the nontuberculous group. Naturally they would have fewer offspring in the F_1 generation. Briefly the method of getting cases is this: The tuberculous subjects in these particular histories were taken at random from the list of active cases

registered in the Tuberculosis Bureau of the Baltimore Health Department. Nontuberculous subjects were taken at random from cases which have passed through the Juvenile Court of Baltimore at one time or another for minor offenses. The individuals are naturally, on the average, younger in the latter case than in the former.

An inexperienced person, not aware of the pitfalls which strew the pathway of the investigator of tuberculosis might think that table 71 told the whole story; that it in short proved the case for the inheritance of the disease. But it will pay us to look a little further into the matter before jumping to this alluring conclusion. Amongst other data which we have collected in this work we have included elaborate and detailed information wherever possible as to the duration and extent of personal contact of individuals in the family history with persons in an active "open" tuberculous condition, whether in the family home or outside. Of course it is impossible to get this information accurately for every individual in a pedigree, and consequently the number of individuals appearing in the following tables is smaller than in table 71. Let us examine the data obtained from such inquiries. The material is exhibited in tables 72 and 73. In this table the headings have the following meanings:

1. *Close contact* means that the enumerated offspring, whether tuberculous or not, were *known* to have been in close and frequent contact with an active case or cases of tuberculosis in their relatives or elsewhere. In case of the tuberculous offspring this contact existed for some time *before* they, the offspring, became themselves tuberculous.

2. *No close contact* means that the enumerated offspring, whether tuberculous or not, were known *not* to have been in close and frequent contact with an active case of tuberculosis among their relatives, and there is no evidence that they were in such contact with active cases among other persons.

Table 72 gives the raw material so far available.

The data of table 72 are summarized in percentage form in table 73. Inasmuch as the number of cases for some of the ancestral combinations is small, we have ventured to combine certain of the rubrics of table 72. It will be noted that even in this small beginning of the contemplated investigation we have included in table 72, 690 individuals for whom we know the contact relations and the tuberculous history of the ancestry for two generations back.

These results are a striking illustration of the caution which must be exercised in drawing conclusions about the inheritance factor in the etiology of tuberculosis. We note:

TABLE 72

Contact relations of individuals in family histories

ANCESTRY	OFFSPRING							
	Nontuberculous		Tuberculous		Suspect		Total	
	Close contact	No close contact	Close contact	No close contact	Close contact	No close contact	Close contact	No close contact
No parent or grandparent tuberculous.	38	300	5	21	1	0	44	321
No parent, one grandparent tuberculous	16	65	5	10	2	0	23	75
One parent, no grandparent tuberculous	46	13	15	1	10	0	71	14
One parent, one grandparent tuberculous	43	19	5	1	0	0	48	20
One parent, two grandparents tuberculous	11	4	2	0	0	0	13	4
One parent, three grandparents tuberculous	2	0	0	0	0	0	2	0
Two parents, no grandparent tuberculous	20	5	13	1	3	0	36	6
Two parents, one grandparent tuberculous	6	3	4	0	0	0	10	3
Totals	182	409	49	34	16	0	247	443

TABLE 73

Percentage of close contact in different ancestral groups

ANCESTRY	PER CENT OF TOTAL OFFSPRING TUBERCULOUS	PER CENT OF TUBERCULOUS OFFSPRING IN CLOSE CONTACT	PER CENT OF NONTUBERCULOUS OFFSPRING IN CLOSE CONTACT
No parent or grandparent tuberculous	7.4	22.2	11.2
No parent, one or more grandparents tuberculous	17.3	41.2	19.8
One parent, and no, one or more grandparents tuberculous	19.8	94.1	73.9
Two parents, and no, or one grandparent tuberculous	38.2	95.2	76.5

1. Where there is no immediate tuberculous ancestry (parents and grandparents nontuberculous) 7.4 per cent of the offspring are actively tuberculous. This probably represents a little less than the normal incidence rate of the disease in the general working class population in Baltimore. Of this 7.4 per cent, however, nearly a fourth (exactly 22.2 per cent) were known to have lived for some time before developing the disease in close contact with an active case or cases of tuberculosis. On the other hand, of the 92.6 per cent of nontuberculous offspring of nontuberculous ancestry only about one-tenth (exactly 11.2 per cent) had been in close contact with an active case. In other words, twice as many of the tuberculous offspring of the nontuberculous ancestry had been in close contact with active, open tuberculosis, as had been the case with the nontuberculous offspring of nontuberculous ancestry.

2. As we pass to the next ancestral rubric, which includes cases where neither parent was tuberculous, but one or more of the grandparents were, it is seen that the percentages all increase. Seventeen and three-tenths per cent of the offspring are tuberculous, but nearly one-half of these tuberculous offspring (exactly 41.2 per cent) were for some time before acquiring the disease in close and intimate contact with an active open case or cases. In the case of the 58.8 per cent of nontuberculous offspring, having the same type of ancestry relative to tuberculosis, only about one-fifth (actually, 19.8 per cent) had been in such close contact with active open cases. Or again we see, just as in the nontuberculous ancestry rubric, that relatively about twice as many of the offspring who developed clinically active tuberculosis had been in close contact with other tuberculous cases as in the case of the nontuberculous offspring of the same type of ancestry.

3. The next line of the table includes the cases of still more pronounced tuberculous ancestry. Here are brought together all of the offspring who had one parent tuberculous, and no, one, or more grandparents tuberculous. In this case, the percentage of the total tuberculous offspring rises only a little above that in the preceding rubric, the increase amounting to only 2.5 per cent, but the contact rates show a relatively enormous difference. Whereas, about one-fifth of the offspring of this type of ancestry themselves show active tuberculosis, nearly all of the individuals who do exhibit the disease (exactly 94.1 per cent) are known to have been in close contact with one or more clinically active cases for some time before they broke down with the disease. Of course, this enormous increase in the percentage is accounted for, to a large degree, by the fact that one parent is tuberculous, and the children have lived with this parent while he or she was actively

tuberculous, but before they (the children) acquired the disease. But whatever the explanation the difficulty in interpreting the data from the standpoint of pure inheritance is obvious. An exactly similar, though not so large, increase is found when we examine the nontuberculous offspring of this same type of ancestry, where one parent, and no, one, or more grandparents are tuberculous. Of the nontuberculous offspring of such ancestry 73.9 per cent have been in close contact for some time with active open cases of tuberculosis.

4. In the last line of the table we have the cases where both parents, and no or one grandparent were tuberculous. Here we find 38.2 per cent of the offspring tuberculous. Of these tuberculous individuals, 95.2 per cent were known to have been for some time in close contact with an active open case or cases of tuberculosis before they exhibited signs of the disease. Of the 61.8 per cent nontuberculous offspring of this same ancestry, about three-fourths (exactly 76.5 per cent) had been in close contact with active open cases to just as great a degree as their tuberculous brothers and sisters without themselves developing the disease.

The results of table 73 are shown graphically in figure 48.

These tables, 72 and 73, demonstrate the difficulty of interpretation which inheres in statistics regarding the inheritance of the tuberculous diathesis. As the amount of tuberculosis in the direct ancestry increases, the amount of tuberculosis in the offspring increases also, but the disturbing fact is that the rate of close contact with open active cases increases enormously more rapidly than does the rate of incidence. In short, we find that where one or both parents are actively tuberculous, practically all the offspring who subsequently develop tuberculosis have been in close, intimate contact with another active case, usually, of course, that of the parent or parents. Instantly those who oppose the view that constitution plays any part in the etiology of disease, and particularly of tuberculosis, will assert that this explains the whole matter—that if the children had not been in contact with the open, active cases, they would not have broken down with the disease. Just possibly they may be right. The case, however, is not simple. Our figures equally show that where one or both of the parents were actively tuberculous, *virtually three-fourths of the nontuberculous offspring have been in just as close contact with active open cases as their brothers and sisters who unfortunately developed the disease.* And it must not be supposed that this high contact percentage can be explained by asserting that the nontuberculous children of tables 72 and 73 are all young children who will subsequently all develop the disease. This is not

true. Their average age is significantly the same as that of the tuberculous offspring.

Since the material is here used for purposes of illustration only, and since the numbers involved must be greatly increased before a final analysis can be undertaken, I shall make no attempt here to go into any further detailed

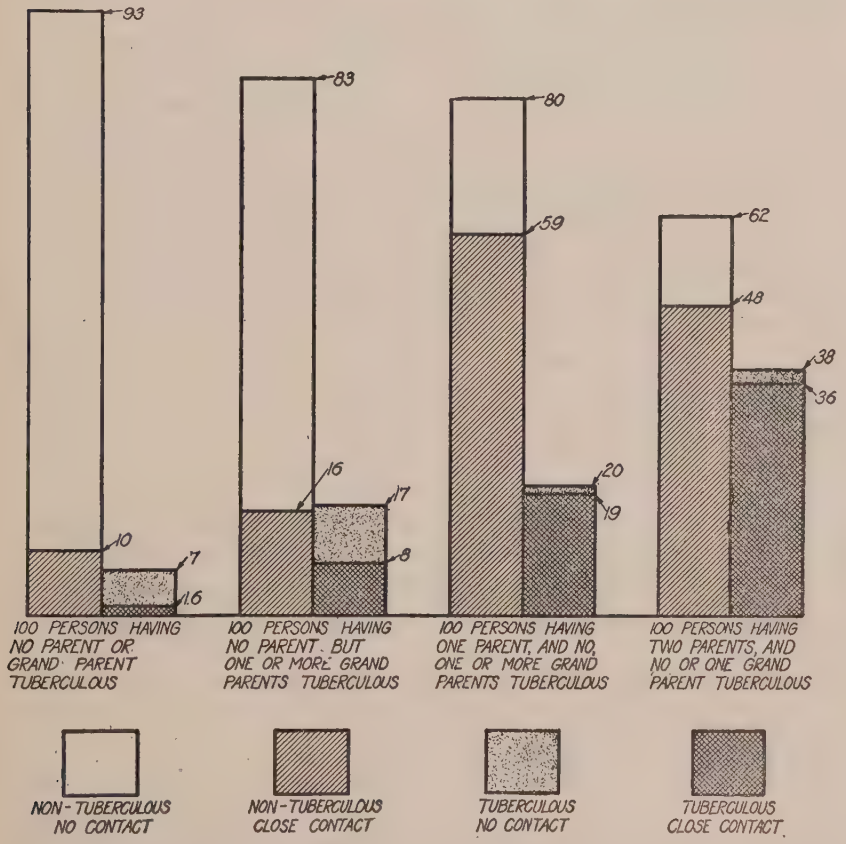

FIG. 48. SHOWING THE CONTACT RELATIONS OF TUBERCULOUS AND NONTUBERCULOUS INDIVIDUALS OF DIFFERENT GRADES OF TUBERCULOUS ANCESTRY, PREPARED FROM DATA OF TABLES 72 AND 73

analysis of this material along lines which obviously suggest themselves. The disturbing thing, and the thing which demonstrates that much further work must be done upon the problem, is that in every case the close contact rate is higher (compare the second with the third column in the table) for the tuberculous offspring than it is for the nontuberculous offspring. In

other words, these figures indicate that familial contact with active open cases is beyond question a factor in determining the incidence rate of clinically active tuberculosis. It appears equally obvious, however, that it certainly does not account for the whole of the increase in the incidence of the disease which we find to occur as the amount of tuberculosis in the immediate direct ancestry increases.

As I have repeatedly stated, these figures are to be regarded as only preliminary, and are introduced here solely for illustration of difficulties of method. Before we shall be satisfied to draw conclusions from them, or to pursue them to their ultimate possibilities in the way of mathematical analysis, we shall need to multiply the numbers many-fold. I do think, however, that the material here presented is sufficient in the first place to justify caution against accepting unreservedly any supposed measurements of the force of inheritance in determining the frequency of breakdown from tuberculosis. In the second place, I think the figures are useful as demonstrating that if the problem is ever to be solved we must obtain data of a much more detailed and accurate character than any of those which, so far as I am aware, have hitherto been used, in attempting to measure the significance of the inheritance factor.

Evidence from the natural history of the disease. We may now pass from a consideration of the direct evidence of the inheritance of the tuberculous diathesis to certain of the lines of indirect evidence, which are not less important in reaching a just conclusion regarding this important problem. A good deal of important indirect evidence may be derived from known facts regarding the natural history of the disease. Let us first in this connection examine what is known about the history of mortality from tuberculosis in old civilizations on the one hand, and newly settled countries on the other hand. Pearson was the first to point out the curious fact that the decline in the rate of mortality from tuberculosis, which in old and long settled countries had been going on at a steady rate for a good many years, ceased to proceed at the same rate almost immediately following the discovery of the bacterial cause of the disease. While to be sure the mortality rate continued to decline, this decline has proceeded at a slower pace since the time of Koch's discovery of the bacillus, and the consequent inauguration of the active "fight against tuberculosis." Pearson first dealt in detail with the corrected mortality rates for England, and compared the phthisis death rate with the general death rate from all causes. He found during the period from 1847 to 1866, which was characterized in the case of the general death rate from all causes by a stability of course, that the death rate from phthisis fell long before the general death rate, and before

what may be termed the period of sanitation. He points out that "this in itself indicates a natural rather than an artificial decay of phthisis." He further showed that during what he terms the "period of sanitation" (1866–1891) the fall in the phthisis death rate was more marked than in the general death rate. During his third or last period (1891–1910), during which time three things happened, namely, the discovery of the tubercle bacillus, the introduction of sanatoria for the treatment of tuberculosis, and the inauguration of the "fight against tuberculosis," the rate of fall in the death rate of phthisis instead of being accelerated was actually retarded.

Examination of the same sort of data from other countries in Europe, and from the part of the United States adjacent to the Atlantic seaboard, where the records run back for a considerable period of years, indicates that these phenomena which Pearson first pointed out for the tuberculosis death rate of England are apparently general and world-wide phenomena, for all biologically similar kinds of population. Owing to lack of space it is impossible to present this material here.

Recently Pearson[8] has brought his discussion of the tuberculosis death rate of England up to date. In 1911 he pointed out that the curves then available seemed to indicate that an actual rise in the phthisis death rate might in the near future be reasonably expected. The indications that this view was correct were still stronger when the returns for 1910 and 1914 were plotted. In the most recent paper he has plotted the figures to and including 1918. Owing to the difficulty in interpreting any vital statistics in a country like England during the war period it is not entirely clear whether the actually observed rise in the rate of tuberculosis mortality which the curves show is to be regarded as a real phenomenon. Pearson's very conservative conclusion in the matter is as follows:

> On the whole it is risky to form a very definite judgment, but, having regard to the female phthisis death-rate and to the percentage of the phthisis death-rate on the general death-rate, war difficulties do not seem to me sufficient to obscure the general trend of our graphs (as indicated before the war), namely, that somewhere about 1915 the fall in the phthisis rate which has been less rapid since 1895 would cease altogether and probably be followed by a *rise*. The next five years will show whether this be true or not. We should expect a fall in the phthisis death-rate immediately, but on the average the value will remain higher than that of 1915.

Strong indirect evidence of the importance of the constitutional factor in the etiology of tuberculosis is afforded by the course of the tuberculosis

[8] Pearson, K., The check to the fall in the phthisis death-rate since the discovery of the tubercle bacillus and the adoption of modern treatment, *Biometrika*, 1919, xii, 374.

death-rate in newly settled countries, where pioneering stock has gone to take advantage of the opportunities of an unexploited land. The course of events in all such countries where it is possible to get figures seems to be something like this. In spite of the fact that the early settlers are for the most part within the age groups where the incidence of tuberculosis is normally heaviest, namely, from say twenty to forty-five years, the tuber-

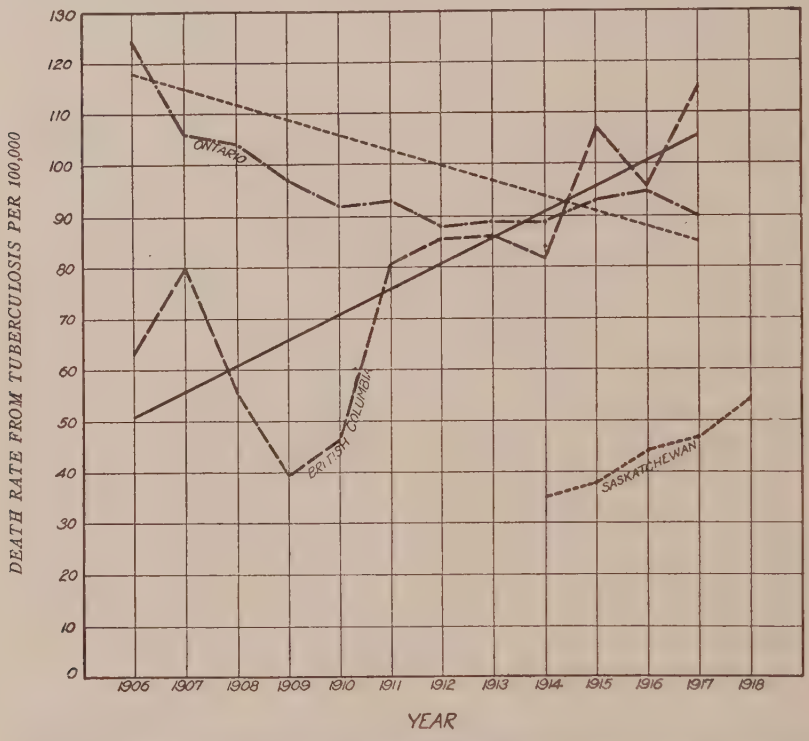

FIG. 49. DIAGRAM SHOWING THE COURSE OF THE DEATH-RATE FROM TUBERCULOSIS IN THE PROVINCES OF BRITISH COLUMBIA, SASKATCHEWAN, AND ONTARIO, CANADA

culosis mortality in these countries is at the outstart very low indeed, and rises, at a more or less rapid rate in different countries, the longer the country has been settled, until finally it reaches what may be regarded as a normal level for the tuberculosis death rate, having regard to the climatic conditions and the racial stocks involved, whatever they may be. As an indication of what happens in such cases figure 49 is inserted, which gives the death rate from tuberculosis for the Provinces of British Columbia and

of Saskatchewan in the Dominion of Canada. For comparison over the same period of time the tuberculosis death rate of the Province of Ontario is inserted. Ontario is, of course, a much longer settled country than either Saskatchewan or British Columbia. Unfortunately, the number of years available for either of the new provinces is small, but the curves given will sufficiently indicate the general trend of affairs.

This diagram shows that in the relatively newly settled provinces of British Columbia and Saskatchewan the tuberculosis death rate is rising.[9] In British Columbia, which has been settled longer (it entered the Canadian Confederation in 1871) than Saskatchewan (which became a province in 1905) the rate has reached what may be regarded as a normal level and is not likely to increase much further. On the other hand the rate in Saskatchewan is extremely low *absolutely*, and is rising rapidly. In both cases the contrast with Ontario, which is inhabited by the same sort of people racially as the other two provinces mentioned, is very great. The Ontario rate is slowly falling, the normal condition in an old, long-settled country.

These results are precisely of the sort which would be expected on biological grounds. Pioneering stock—the sort of people who adventure to settle new countries—is made up mainly of constitutionally sound and vigorous people. They are of the age most liable to tuberculosis, but they are constitutionally not the kind of persons who have tuberculosis. Consequently, we find the death rate from tuberculosis in the earlier years of such settlement of new country, very low. But it steadily rises. As the new land is opened up by the pioneers and living is proved by them not to be too hazardous a business, people of weaker stocks follow them into the country. The death rate from tuberculosis in consequence begins to rise and continues steadily to do so till a stable absolute level is reached corresponding to the normal for the climatic conditions and racial stocks involved.

The phenomenon which is here illustrated by the data from these three Canadian provinces appears to be general for new countries as compared with those of long settlement.

Evidence from differential racial incidence in the same environment. It is a well known fact that different races and peoples exhibit statistically widely different rates of mortality from tuberculosis. I do not now refer to any comparison of uncivilized races initially exposed to the tubercle bacillus,

[9] In order to show more clearly the trend of the mortality, straight lines have been fitted to two of the curves in figure 49. The equation to the British Columbia curve is $y = 45.9 + 4.9x$, where y denotes death rate and x time. The Ontario curve is $y = 110.4 - 2.1x$.

TABLE 74

Comparative mortality from pulmonary tuberculosis of different race stocks in New York and Pennsylvania, 1910

SEX AND AGE PERIOD	DEATH RATES PER 100,000 LIVING AT SAME AGES, OF PERSONS BORN IN													
	Austria-Hungary		Russia		Italy		Germany		England, Scotland and Wales		Ireland		United States (whites only)	
	Pennsylvania	New York	Pennsylvania	New York	Pennsylvania	New York	Pennsylvania	New York	Pennsylvania	New York	Pennsylvania	New York	Pennsylvania	New York
Males														
All ages	118.0	166.0	107.4	114.7	81.5	112.1	194.9	267.4	150.2	215.2	342.8	589.3	105.1	170.9
Under 10	13.8	16.7	15.4	23.5	19.9	8.3	147.9			23.4			23.3	23.4
10–14	35.5	14.1		11.7	23.0	15.1		52.8		36.9			11.2	10.2
15–19	55.4	102.4	69.6	60.5	43.4	106.8		90.8	120.3	128.7	428.0	312.3	61.1	101.5
20–24	106.5	93.1	99.6	96.4	86.5	140.4	211.3	45.9	59.3	71.5	127.7	327.3	147.6	216.3
25–44	110.6	177.2	105.9	117.1	71.2	102.0	198.2	252.5	151.5	240.5	375.8	662.9	185.2	352.0
45–64	264.2	302.5	225.1	246.4	154.3	172.9	230.3	350.0	165.3	268.9	408.8	682.1	174.1	262.1
65–84	242.7	247.9	148.7	182.4	631.8	208.5	140.6	211.3	233.5	210.3	206.1	329.3	189.3	161.4
85 and over							180.2	210.7				357.6	60.0	69.3
Females														
All ages	130.2	102.6	91.7	74.6	102.2	160.1	90.4	115.3	133.2	123.3	201.2	276.1	98.8	109.6
Under 10	27.4		31.3		42.0	34.0		51.7	41.6	23.2			19.1	20.6
10–14	17.3	13.7		5.7		89.1						138.1	19.3	27.1
15–19	74.2	49.6	70.9	28.0	102.0	220.6	39.1	76.9	84.4	64.0	63.6	185.6	91.5	111.5
20–24	141.2	87.9	92.6	88.7	153.6	247.7	128.0	158.2	80.4	121.5	121.6	167.7	162.3	186.3
25–44	152.7	137.2	108.0	104.7	114.8	159.3	106.2	125.1	166.9	165.4	235.3	353.4	172.8	193.3
45–64	162.3	122.9	124.6	80.8	77.7	123.3	85.4	109.7	112.2	92.1	220.7	250.1	108.9	111.1
65–84	215.7	142.1	178.7	94.3	217.3	55.4	72.9	106.3	153.7	139.3	140.9	192.1	178.7	137.9
85 and over						793.7	156.7	80.3	314.5	164.5	148.6		100.9	32.6

with civilized races long habituated to its presence. It is well known that the ravages of this disease, as well as many others, are terrific among people meeting it for the first time. Such facts have little if any evidential bearing upon our present problem. There is, however, another set of facts which appears to be of considerable significance in indicating that genetic constitution plays an important part in the etiology of the disease. This is the widely different rates of fatal breakdown from tuberculosis among different racial stocks living in the same general environment. The most recent data upon the subject are furnished by Dublin and Baker[10] in a notable piece of biostatistical research.

Table 74 is compiled from their data.

From this table it is evident that different racial stocks, living under the same climatic conditions, exhibit wide differences in their mortality from tuberculosis. Contrast the Italian, with a male mortality at all ages from tuberculosis of 81.5 per 100,000 in Pennsylvania, with that of Irish males in the same state, which reaches the value of 342.8 per 100,000. For the same two race stocks the females show in the same state mortality rates of 102.2 and 201.2 per 100,000 respectively. It is evident that the Irish react to the same environment in a totally different way than do the Italians in respect of tuberculosis. The relatively great racial susceptibility of the Irish to this disease is well-known. It appears wherever vital statistics are recorded.

There are many interesting points of detail which might be discussed in connection with table 74, but space is lacking here. I merely insert the table as a standing demonstration of the broad general fact that there are wide differences in respect of mortality from tuberculosis among different race stocks living in the same general environment. These differences are larger in amount than can reasonably be explained by any differences which may be supposed to exist, or do in fact exist, between the different stocks in respect of habits of living, social or economic status, or other environmental factors.

CONCLUSION

In so brief a space it is wholly impossible to marshal more than the smallest fraction of the pertinent evidence which bears upon this problem. All that has been attempted here, and if that has been accomplished I am satisfied, is to demonstrate, in the first place, that the problem of the

[10] Dublin, L. I., and Baker, G. W., The mortality of race stocks in Pennsylvania and New York, 1910, *Quart. Publ. Amer. Stat. Assoc.*, 1920, xvii, 13.

causation of breakdown with clinically active tuberculosis, is a problem of tremendous complexity and difficulty, towards the solution of which only the merest beginning has been made, and in the second place, that the inherited constitution of the individual is a factor in the problem of a great deal more than the negligible importance which some in high positions would accord it. I realize as clearly as my critics, potential and actual, possibly can that the evidence which I have presented here, as well as practically all of the other evidence now existing regarding the rôle of the constitutional factor in tuberculosis, is capable of other interpretation than the one here suggested. I personally think that these other possible interpretations of the facts I have set forth are extremely improbable, and that the one I have suggested is probably the correct one. I cannot as yet, however, *prove* the absolute correctness of my faith. But I also perceive with equal clearness that the environmentalists' case, the infectionists' case, and all the rest are on no better footing. The plain fact is that we are densely ignorant of the relative influence of the several factors which may be concerned in the etiology of tuberculosis. The task before the investigator is to devise and accomplish a real measurement of the relative importance of the hereditary and environmental factors in the tuberculosis problem.

With the closing remarks of Dr. William Bulloch, in the discussion following the reading of the notable paper by himself and Dr. Major Greenwood before the Royal Society of Medicine, I find myself in complete agreement. Doctor Bulloch said that the object in presenting the paper was:

> to enter a protest against the wild statements now being made in the lay and medical press, that the whole problem of phthisis was one of infection. Medical history showed that in tuberculosis, as also in the case of other diseases, the most extreme views were taken, not by those who had contributed the actual advancement in knowledge, but by those whose business it was to apply those advancements for the needs of the public. There were a large number of well-ascertained facts which were not entirely explicable on the doctrine that disposition was not an important factor in the genesis of the disease, and that before rigorous measures were applied on a wide scale the actual facts should be ascertained. He did not agree that public health authorities must always "do something." This "doing something" should be put a stop to until there was a reasonable supposition that it was going to achieve its end. He did not wish it to be understood that the tubercle bacillus was not a potent factor. What he did refuse to believe was that it was the only factor. He considered that the disposition, the power of the individual to resist the aggressive inroads of the bacillus, was greater than many people held at the present day.

Finally, I wish also to quote the closing words of a notable paper by Dr. S. Lyle Cummins.[11] He says: "These considerations indicate that exhaustive research is still necessary in order that efforts at the control of tuberculosis may be directed upon effective lines." With this opinion I most heartily agree.

[11] Cummins, S. L., Tuberculosis in primitive tribes and its bearing on the tuberculosis of civilized communities, *Internat. Jour. Pub. Health*, 1920, i, 137.

CHAPTER XI

The Influence of Physical Activity upon Mortality[1]

THE PROBLEM

In a recent book Pearl[2] suggested that (Pages 211 and 212):

the manner in which the environmental forces (of sub-lethal intensity, of course) chiefly act in determining duration of life, appears to be by changing the rate of metabolism of the individual. Furthermore one would suggest, on this view, that what heredity does in relation to duration of life is chiefly to determine, within fairly narrow limits, the total energy output which the individual can exhibit in its life time. This limitation is directly brought about presumably through two general factors: viz., (*a*) the kind or quality of material of which this particular vital machine is built, and (*b*) the manner in which the parts are put together or assembled. . . . . There is some direct experimental evidence, small in amount to be sure, but exact and pertinent, to the effect that the duration of life of an animal stands in inverse relation to the total amount of its metabolic activity, or put in other words, to the work, in the sense of theoretical mechanics, that it as a machine does during its life.

He then went on to say, further on (page 216), that:

It would seem, at first thought, that one should be able to test the theory here suggested, that rate of energy expenditure in the business of living is negatively correlated with the total duration of life, by an examination of the mortality rates for persons in different occupations as set forth, for example, in the well known paper of Bertillon.[3] When one endeavors to make such a test, however, he is at once confronted with a series of difficulties which presently convince him that the project is virtually an impossible one, if he wishes critical results. In the first place, mean age at death will not do as a criterion, because of the great differences in the age distributions of those engaged in different occupations. This point has lately been thoroughly discussed by Collis and Greenwood,[4] in their book *The Health of the Industrial Worker*. Indeed, their whole treatment of the problem of occupational mortality is by far the most sound and critical which the present writer has yet seen. One must deal with age and sex specific death rates, or mortality indices based upon them.

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[1] By Raymond Pearl and Paul R. Hawley. The material in this chapter has not previously been published.

[2] Pearl, R., *The Biology of Death*, Philadelphia (Lippincott), 1922.

[3] Bertillon, J., Morbidity and mortality according to occupation, *Jour. Roy. Stat. Soc.*, Vol. 55, pp. 559–600, 1892.

[4] Collis, E. L., and Greenwood, M., *The Health of the Industrial Worker*, London. 1921, 450 pp.

In the second place, there are specific hazards, direct or indirect, in various occupations, quite apart from any question of energy expenditure involved in the case. These hazards will, obviously, tend to obscure any direct effects of the energy relations involved.

In the third place, we have only the merest suggestion of quantitatively accurate knowledge as to the average energy output involved in different trades and occupations.

Since the above was written certain extremely valuable detailed statistics on the mortality of occupied males have been published from the office of the Registrar-General of England and Wales.[5] These data fulfill most of the requirements set forth above. It is the purpose of this chapter to present an analysis of these figures undertaken to see what light they might shed upon the theory suggested in the first quotation given above.

The nature of the material contained in the Report from the Registrar-General's office may be indicated as follows. The figures cover the deaths in the years 1910 to 1912 inclusive, together with the corresponding populations derived from the Census of 2/3 April, 1911. The raw data are given in age groups 15 to 19, 20 to 24, 25 to 34, 35 to 44, 45 to 54, 55 to 64, 65 to 74, and 75 and upwards; under 27 separate cause rubrics, and all causes, for each of 132 main occupational groups (and some subdivisions of these not taken account of in the present study).

Regarding the occupational classification the following statement is made in the introductory text of the Report (page vii):

The classification is, on the whole, rather of an industrial than of a strictly occupational nature, and thus obscures in many instances the full effect of dangerous processes or operations. It deals with such groups, for instance, as men making cutlery, or motor chassis, or pottery; but it does not distinguish how many of these men are grinders, or sand blast operators, or lead glaze workers, and thus precludes any estimation of the real risk to life involved in those processes by themselves. Clearly the inclusion of such workers in a larger group with many workers engaged upon less harmful processes is bound to show a mortality reflecting the diluted average risk to which the whole group is exposed, whereas the special risk in the more dangerous process would be thrown up into sharp relief by the separate treatment of the workers engaged upon it.

The plan of the following study may be stated in the following way: Suppose it were possible to arrange the 132 occupations given in table 75 *infra* in order in a list, such that the occupation which, on the average, required the *least* expenditure of physical energy in its pursuit over the whole life span, stood *first* in the list; that occupation requiring the next higher expenditure of physical energy stood next; and so on to the last occupation

[5] Mortality of men in certain occupations in the three years 1910, 1911, and 1912. Supplement to the 75th Annual Report of the Registrar-General for England and Wales. London (H. M. Stationery Office), not dated, but actually 1923, pp. xxiv + 100.

in the list, which would be that one requiring the *greatest* expenditure of energy. How then would the age specific death rates set against each of these occupations arrange themselves? Would the first occupation in the list exhibit the lowest specific death-rates, and the last the highest, with the occupations in between having their specific death-rates in orderly progression upwards?

This is the *ideal* statement of our problem. Actually this ideal is, of course, unattainable practically for several reasons, of which perhaps the most important is that it is wholly impossible to arrange the 132 occupations in an *exact* scale of ascending order of average expenditure of physical energy incident to this occupation. So then the first step towards practical work upon the problem is to get the occupations grouped into a much smaller number of rubrics than 132, in the hope that by taking broad classes we can reasonably arrange the material in a scale of ascending physical energy expenditure. Furthermore, since we are dealing in this matter with unmeasured phenomena, where the placing of a particular occupation in the list will represent simply some person's *estimate* of the physical difficulty of the tasks involved in that occupation, it will be advisable to take the average of several such estimates.

Suppose all this to have been done. There will still remain some other points which must be taken into account. In the first place purely accidental deaths must be eliminated from the reckoning, because the accidental termination of a particular life as a result of the sudden impact of an automobile against the individual's *corpus*, for example, has no relation to whether he was a hard working coal heaver or a delicate curate whose most strenuous physical labor had been an occasional hard fought game of croquet. This elimination is not especially difficult, because one of the cause rubrics of the Report is "Accident," and these deaths may be at once subtracted out of the total. The accidental deaths so taken out will include those incident to the hazards of the occupation. But, from the standpoint of the present inquiry this is proper and just, because what we are concerned with here is *not* occupation *per se* or its mortality, but with occupation as an index, rough perhaps but still generally reliable, of the physical strenuousness of the life lived by different groups of individuals. The fact that accidental external violence is more likely to kill stevedores than clergymen because of the environmental circumstances of their respective occupations, has no bearing upon, or pertinent relation to, the fact that stevedores do physically harder work than clergymen.

This same reasoning will lead us further to subtract out of the total mortality of each occupation the non-accidental (in the sense of the sta-

tistical classifier) deaths known to be directly due to the hazards of the occupation. To do this offers more practical difficulties, for obvious reasons. But fortunately the statistics of the report are so arranged as to make this in some degree possible. Thus, in the case of occupation rubric 29 (dock laborers, wharf laborers) 2 deaths from anthrax acquired in the course of the work at this occupation are deducted. In the same way from the total mortality in rubric 49 (iron and steel manufacture) 3 deaths from occupational lead poisoning are deducted. It is, however, quite certain that the officially classified statistical returns do not permit the determination and deduction of anything like all of the mortality which in real truth should be charged directly to the hazards of the occupation *per se*. This is evident on general principles. It is specifically demonstrated in a number of instances in the able paper of Ward.[6]

It should be clearly understood that we have deducted deaths due to occupational hazards only where the tables of the original report specifically stated that they belonged in this category. In other words the responsibility of the decision that particular deaths were of this sort is not ours.

Two other factors not so readily corrected or allowed for may influence the results. These are selective recruiting in certain occupations, and shifting about of the individual among various occupations. By the first of these is meant the somewhat automatic tendency of certain occupations to attract preponderantly individuals of superior physical fitness. Naturally such individuals will, by virtue of their inherited constitution, have a superior expectation of life. Hence the true results in respect of mortality which might otherwise flow from the physical energy expenditure relations in this occupation, will tend to be altered or obscured. The discussion of these points in the introductory text of the report (pages ii and iii) is worth quoting in detail, as it will enable the reader to judge for himself what their significance is.

> Something must also be said with regard to the other term in the equation—the man himself. Even if the environment of all men were the same, the men themselves would differ widely and would react differently to their environments. If the risks to life, both direct and indirect, of all occupations were equal, there might still be much difference between the mortalities of different bodies of workers. Some occupations are followed by older, some by younger men; and difference in ages can, of course, be allowed for. But other differences in regard to what may be called the health capital invested in the several occupations cannot be similarly dealt with; and the possibility of such differences and the

[6] Ward, L., The effect, as shown by statistics, of British statutory regulations directed to the improvement of the hygienic conditions of industrial occupations, *Jour. Roy. Stat. Soc.*, vol. 68, pp. 435–518, 1905.

extent to which they affect any conclusions must constantly be borne in mind in the interpretation of the figures.

Were it the case that the distribution of the strong and the weak among different occupations is governed solely by personal caprice, it might be reasonable to hold that inequalities would level out and deserve no special allowance. But it is apparent that there are selective tendencies at work. In the first place, some occupations are ordinarily recruited from sections or classes of the community which exhibit a special health standard of their own: agricultural workers, for example, are usually recruited from the more healthy rural population. But, generally speaking, the selection operates by reference to the physical aptitude of the individual worker, whether it takes effect through his deliberate choice or change of occupation, or through his unwilling exclusion or expulsion. Some occupations may repel, while others attract, the unfit at the age of starting work, and, conversely, some occupations may be of necessity recruited from men of supernormal physical condition. Again, it may well be that the more exacting occupations are continuously giving rise to a process of the selective discharge and transfer to other occupations of those persons who have proved unequal to the strain imposed upon them. This is especially likely to occur during the later years of life; and would, of course, result in the understatement of the mortality of the more arduous occupations at the expense of overstatement in the case of those in which refuge is taken.

For example, it will appear from the tables that engine drivers (group 19) and motor-car drivers (group 24) are subject to abnormally low death-rates. But before it can be inferred that engine driving and motor-car driving are subject to a relatively light mortality risk it must first be considered whether the low mortality is not due to the selective recruitment of men of a high standard of fitness; or, alternatively, whether there is not a selective discharge from those occupations of men whose health has deteriorated under the strain of their work, and who thus, passing out of observation, relieve the occupations of some part of the mortality which should statistically be debited to them.

It may be urged, on the other hand, that under the influence of trade union rules labour has tended to be less fluid, and that within many skilled occupations or groups of closely allied skilled occupations there is a measure of latitude between the more and the less exacting work which admits of a process of accomodation whereby it is made possible for a man to be retained notwithstanding loss of fitness.

It may thus be the case that, apart from certain special instances, the element of selective transfer does not operate to any material extent save in the case of the younger men who have not made a final choice of occupation, and as between the skilled occupations and the mass of unskilled labour at the bottom of each industry. There can be little doubt that in both these cases the tendency to selective transfer is strongly operative. It will be seen from table III that the occupation having the worst mortality experience is that of the general laborer. In this group (amounting to 321,093) the deaths in excess of the expected mortality (28,723) outnumber the aggregate excess deaths for all other occupational groups similarly exceeding expectation. They also outnumber the net excess deaths (28,185) in the larger group of labourers (over a million) of which the general laborer group forms part. Doubtless the low economic status of the class would conduce to a high mortality rate; and equally doubtless a part of the excess is certainly due to causes indicated later as arising out of the particular material employed in the compilation of these tables. But it is difficult to escape the conclusion that a portion of the excess is due to the overloading of the class with individuals who, through diminished physical or mental health, have drifted into it through inability to follow a former occupation. It is

to unskilled and ill-defined occupations that such transfers might specially be expected to take place; and the inference is supported in the case of this group to some extent by the rather large proportion of older men recorded within it.

The practical conclusion regarding this matter is, we think, that if any results which we may get from our examination of the data are small in amount, or inconstant in direction, the existence of these uncorrected factors of selection and shifting will make it impossible to draw any conclusions. If, on the other hand, our results are large in amount and consistent in trend, it may be safely concluded that the uncorrected factors are not important enough to alter significantly the general trend of the data.

There is, however, another and more serious source of error in the material, which arises from the fact that in the higher age groups the occupational record of deaths is far more nearly exhaustive and correct than the occupational record of those exposed to risk. This is made clear by the following figures for "unoccupied males" (Report, page iv):

	AGE				
	15 to 25	25 to 45	45 to 65	65 to end	Total
Actual deaths	5,191	1,995	2,206	3,897	13,289
Expected deaths in the unoccupied population according to mortality rates of "all males"	1,141	883	3,340	28,668	34,032
Excess (+) or deficiency (−) of actual over expected	+4,050	+1,112	−1,134	−24,771	−20,743
Ratio of actual to expected	4.55	2.26	0.66	0.14	0.39

The mortality of this class is thus four and one-half times the normal under age 25, and this diminishes with advancing age until it becomes the ridiculously low rate of one-seventh normal for ages over 65.

This error, which leads to a definite overstatement of the mortality of occupied males at ages 65 on, is probably correlated to some unknown and undeterminable degree with strenuousness of occupation. The discrepancy below age 25 may be disregarded in the present inquiry because the number of unoccupied is small compared with total males. But above 65 the absolute numbers involved form a sensible fraction of the total.

The practical conclusion is that in this study no reliance can be placed on death rates at ages 65 and over as absolute figures, and their comparative significance in the way they are used in the present study is, at the best, uncertain, because of the possible correlation of the errors with strenuousness of occupation. We shall table the rates at these ages for the sake

of completeness, but in every case mark them in some distinctive manner, so that the reader may not be misled into attaching too much significance to them.

The point may be raised that the same error occurs in some degree after age 45. But before age 65 it must be negligible in amount, so far as concerns the large differences in mortality which will be shown between the groups of occupations set up later in this study. That this error is negligible up to age 65 is furthermore indicated by the fact that the Registrar-General's office in the Report calculates and publishes comparative mortality figures for the single occupations up to age 65.

PRELIMINARY TREATMENT OF THE RAW DATA

The list of occupations as it appears in the original Report is given in table 75.

Photostat copies of table 75 were prepared, and to each set was attached an instruction sheet, reading as follows:

INSTRUCTIONS FOR THE CLASSIFICATION OF OCCUPATIONS

The object of this classification is to take the list of occupations, already separated by Pearl and Hawley into two broad groups of chiefly indoor and chiefly outdoor occupations, and to arrange the several occupations in each of these broad groups into five sub-groups with respect to the relative expenditure of *physical energy* which in your opinion is incident to the included occupations.

These sub-groups are to be arranged in *ascending* order of their physical energy requirements. Group I will thus include those occupations requiring the least expenditure of physical energy. Group V will include those requiring the greatest output of physical energy.

No consideration is to be given to the mental strain or to the accidental hazards incident to the occupation. For example, in arriving at the relative standing of airplane pilots and book-keepers, only the factor of the expenditure of physical energy required by each occupation is to be considered.

It is suggested that the most satisfactory method of making this classification is to form the idea of an average in the indoor group as a whole and the outdoor group as a whole, entering the occupations which you regard as about average as in the middle sub-group III. For example, if it is your opinion that the occupation of typist represents about the average expenditure of physical energy among the indoor occupations, this occupation should be entered in sub-group III under indoor occupations. If an artist's model is believed to expend less physical energy, this occupation should be entered in sub-group II. If a coal heaver expends a maximum amount of energy, this occupation would fall in sub-group V; etc. A similar procedure may be followed in the arrangement of the outdoor occupations.

In the tabulation of the classification on the enclosed blank tables, the reference *numbers* of the occupations which appear in the extreme left hand column may be used instead of the cumbersome names of the occupations.

Those occupations *only* whose numbers are encircled with red are to be classed "chiefly outdoor." Please follow this instruction exactly.

TABLE 75

List of occupational groups as given in the original Report (1910–1912) and the occupational headings of the Census comprised in each group (from Table II of Report)

REFERENCE NUMBER	OCCUPATIONAL GROUPS, 1910–12	OCCUPATIONAL HEADINGS IN CENSUS REPORT
	Title	Title
1	Civil Service (Officers and Clerks)	Post Office—Telegraphists, Telephone Operators. Other Post Office Officers and Clerks Other Civil Service Officers and Clerks
2	Civil Service (Messengers, etc.)	Postmen Post Office Messengers, etc. Other Civil Service Messengers, etc.
3	Clergymen, Priests, Ministers	Clergymen (Established Church) Roman Catholic Priests Ministers, Priests, of other religious bodies
4	Barristers, Solicitors	Barristers Solicitors
5	Law Clerks	Law Clerks
6	Physicians, Surgeons, Registered Practitioners	Physicians, Surgeons, Registered Practitioners
7	Schoolmasters, Teachers	Schoolmasters, Teachers, Professors, Lecturers
8	Artists, Engravers, Sculptors, Architects.	Painters, Sculptors, Artists Architects Engravers
9	Photographers	Photographers
10	Musicians, Music Masters	Musicians, Music Masters, Singers
11	Domestic Indoor Servants (not in Hotels, Lodging or Eating Houses)	"Other" Domestic Indoor Servants
12	Gamekeepers	Gamekeepers
13	Commercial Travellers	Commercial Travellers
14	Commercial Clerks	Commercial or Business Clerks
15	Bankers; Bank—Officials, Clerks	Bankers; Bank—Officials, Clerks
16	Insurance—Officials, Clerks	Life, House, Ship, etc., Insurance—Officials, Clerks, etc.
17	Insurance Agents	Insurance Agents
18	Railway—Officials, Clerks	Railway—Officials, Clerks
19	Railway Engine—Drivers, Stokers, Cleaners	Railway Engine—Drivers, Stokers, Cleaners
20	Railway—Guards, Porters, Pointsmen, Signalmen, etc.	Railway Ticket—Examiners, Collectors, Checkers Railway Guards Signalmen Pointsmen, Level Crossing Men Railway Porters Other Railway Servants

TABLE 75—*Continued*

REFERENCE NUMBER	OCCUPATIONAL GROUPS, 1910–12	OCCUPATIONAL HEADINGS IN CENSUS REPORT
	Title	Title
21	Platelayers, Gangers, Packers	Platelayers, Gangers, Packers
22	Railway Labourers	Railway Labourers (not Railway Contractors' Labourers).
23	Coach, Cab, Omnibus—Service; Grooms, etc.	Livery Stable Keepers; Coach, Cab—Proprietors. Omnibus Service— Horse Drivers Motor Drivers Conductors Others Domestic—Coachmen, Grooms Coachmen (not Domestic); Cabmen Horsekeepers, Grooms, Stablemen (not domestic).
23*a*	Domestic—Coachmen, Grooms	Domestic—Coachmen, Grooms
24	Motor Car, Motor Van—Drivers	Domestic—Motor Car Drivers, Motor Car Attendants Motor Car Drivers (not Domestic); Motor Cab Drivers Motor Van, etc., Drivers
25	Carmen, Carriers, etc.	Carmen, Carriers, Carters, Wagoners (not Farm). Van, etc.—Guards, Boys Others connected with carrying or cartage
26	Tramway Service	Tramway Service—Drivers Conductors Others
27	Seamen, etc., Merchant Service	Merchant Service; Seamen Navigating Department Engineering Department Cooks, Stewards and Others (Subsidiary Services) Pilots, Boatmen on Seas
28	Bargemen, Lightermen, Watermen	Bargemen, Lightermen, Watermen
29	Dock Labourers, Wharf Labourers	Dock Labourers, Wharf Labourers
30	Coalheavers	Coalheavers; Coal—Porters, Labourers
31	Messengers, Porters, Watchmen (not Railway or Government)	Messengers, Porters, Watchmen (not Railway or Government)
32	Farmers, Graziers, Farmers' Sons, etc.	Farmers, Graziers Farmers', Graziers'—Sons or other Relatives assisting in the work of the farm

TABLE 75—*Continued*

REFERENCE NUMBER	OCCUPATIONAL GROUPS, 1910–12	OCCUPATIONAL HEADINGS IN CENSUS REPORT
	Title	Title
33	Agricultural Labourers, Farm Servants	Agricultural Labourers, Farm Servants
		in charge of Cattle
		in charge of Horses
		not otherwise distinguished
34	Gardeners, Nurserymen, Seedsmen	Domestic Gardeners
		Nurserymen, Seedsmen, Florists
		Market Gardeners (including Labourers)
		Other Gardeners (not Domestic)
35	Fishermen	Fishermen
36	Coal Miners	Coal and Shale Mine
		Workers at the Face
		Other workers below ground
		Workers above ground
37	Iron—Miners, Quarriers	Iron—Miners, Quarriers
38	Tin Miners	Tin Miners
39	Lead Miners	Lead Miners
40	Coke Burners	Coke Burners
41	Patent Fuel Manufacture	Patent Fuel Manufacture
42	Stone—Getters and Dressers; Masons	Stone—Miners, Quarriers
		Stone—Cutters, Dressers
		Masons
		Masons' Labourers
		Architectural, Monumental Carvers, Sculptors
		Monumental Masons
43	Slate—Quarriers, Workers	Slate—Miners, Quarriers
		Slate Workers
44	Coal, Coke—Merchants, Dealers	Coal, Coke—Merchants, Dealers
45	Engine, Machine, Boiler—Makers, Fitters; Millwrights	Patternmakers
		Millwrights
		Erectors, Fitters, Turners
		Erectors', Fitters', Turners'—Labourers
		Metal Machinists
		Labourers (undefined) in Engineering Works
		Boiler Makers
		Other or Undefined Workers in Engineering and Machine Making
		in. Textile Machinery, Fittings, etc.
		Others

TABLE 75—*Continued*

REFERENCE NUMBER	OCCUPATIONAL GROUPS, 1910–12	OCCUPATIONAL HEADINGS IN CENSUS REPORT
	Title	Title
45*a*	Patternmakers	Patternmakers
45*b*	Millwrights, Fitters, Turners	Millwrights
		Erectors, Fitters, Turners
45*c*	Boiler Makers	Boiler Makers
46	Electrical Apparatus Makers; Electricians, Electrical Fitters	Electrical Cable Manufacture
		Electric Lamp Manufacture
		Other Electrical Apparatus Makers; Electrical Fitters
		Electricians (undefined)
47	Tool, Scissors, File, Saw, Needle Makers	Tool Makers
		File Makers
		Saw Makers
		Cutlers; Scissors Makers
		Needle, Pin—Makers
47*a*	File Makers	File Makers
47*b*	Cutlers; Scissors Makers	Cutlers; Scissors Makers
48	Gunsmiths	Gunsmiths, Gun Manufacturers
49	Iron and Steel Manufacture; Iron Goods Makers	Pig Iron Manufacture (Blast Furnaces)
		Puddling Furnaces; Iron and Steel Rolling Mills
		Tube Manufacture
		Steel—Manufacture, Smelting, Founding
		Galvanized Sheet Manufacture
		Ironfounders
		Stove, Grate, Range, Fire Iron—Makers
		Bedstead Makers (Iron or Brass)
		Other Iron Goods Makers
		Iron Workers (undefined)
49*a*	Ironfounders	Ironfounders
49*b*	Stove, Grate, Bedstead—Makers	Stove, Grate, Range, Fire Iron—Makers
		Bedstead Makers (Iron or Brass)
50	Anchor, Chain—Manufacture; Blacksmiths	Blacksmiths, Strikers
		Anchor, Chain—Manufacture
51	Wire—Drawers, Makers, Workers, Weavers	Wire—Drawers, Makers, Weavers, Workers
52	Nail, Bolt, Lock, Key—Makers	Nail Manufacture
		Bolt, Nut, Rivet, Screw, Staple—Makers
		Lock, Key—Makers
53	Tinplate Manufacture	Tinplate Manufacture

TABLE 75—*Continued*

REFERENCE NUMBER	OCCUPATIONAL GROUPS, 1910–12	OCCUPATIONAL HEADINGS IN CENSUS REPORT
	Title	Title
54	Zinc Manufacture	Zinc Manufacture
55	Copper Manufacture; Copper Workers, Coppersmiths	Copper Manufacture Coppersmiths Copper Workers
55*a*	Copper Manufacture (Carmarthenshire and Glamorganshire)	Copper Manufacture
55*b*	Coppersmiths, Copper Workers (in a group of counties where there is no copper smelting)	Coopersmiths Copper Workers
56	Tinplate Goods Makers	Tinplate Goods Makers
57	Lead Manufacture; Leaden Goods Makers	Lead Manufacture Leaden Goods Makers Dye, Paint, Ink, Blacking—Manufacture (part of)
58	Brass, Bronze—Manufacturers, Founders, Finishers, Workers	Brass, Bronze—Manufacture Brassfounders Brass Finishers Gas Fittings Makers Lamp, Lantern, Candlestick—Makers Brass, Bronze—Workers
58*a*	Brass, Bronze—Manufacture; Brassfounders	Brass, Bronze—Manufacture Brassfounders
58*b*	Brass, Bronze—Workers	Brass Finishers Gas Fittings Makers Lamp, Lantern, Candlestick—Makers Brass, Bronze—Workers
59	Shipbuilding	Ship—Platers, Rivetters, etc. Other Workers in Iron Shipwrights Ship—Other Workers in Wood Painters Shipyard Labourers (undefined) Others in Ship and Boat Building
59*a*	Shipbuilding—Workers in Iron	Ship—Platers, Rivetters, etc. Other Workers in Iron
59*b*	Shipbuilding—Workers in Wood	Shipwrights Ship—Other Workers in Wood
60	Cycle Makers	Cycle Makers
61	Coach, Motor, Railway, Carriage, Tram Car, etc.—Makers	Railway—Coach, Wagon Makers Tram Car Makers Motor Car Chassis Makers; Motor Car Mechanics Motor Car Body Makers Coach, Carriage—Makers Others in Construction of Vehicles

TABLE 75—*Continued*

REFERENCE NUMBER	OCCUPATIONAL GROUPS, 1910–12	OCCUPATIONAL HEADINGS IN CENSUS REPORT
	Title	Title
62	Wheelwrights	Wheelwrights
63	Ironmongers	Ironmongers; Hardware—Dealers, Merchants
64	Watch, Clock—Makers; Jewellers	Goldsmiths, Silversmiths, Jewellers Lapidaries and Other Workers Watchmakers, Clockmakers Dealers in Precious Metals, Jewellery and Watches
64*a*	Jewellers (in Staffordshire, Warwickshire and Worcestershire)	Goldsmiths, Silversmiths, Jewellers
65	Scientific, Weighing and General Instrument Makers	Scientific Instrument Makers; Opticians Photographic Apparatus Makers Weighing and Measuring Apparatus Makers Surgical and Dental Instrument and Apparatus Makers
66	Piano, Organ—Makers	Piano, Organ—Makers
67	Builders	Builders
68	Carpenters, Joiners	Carpenters, Joiners
69	Bricklayers	Bricklayers
70	Slaters, Tilers	Slaters, Tilers
71	Paperhangers, Plasterers, Whitewashers	Plasterers Paperhangers, Whitewashers
72	Painters, Decorators	Painters, Decorators Ship Painters
73	Plumbers, Glaziers, Gasfitters	Glaziers Plumbers Gasfitters Locksmiths, Bellhangers
74	Cabinet Makers, French Polishers, Upholsterers	Cabinet Makers French Polishers Upholsterers
75	Furniture, etc., Dealers	Furniture, etc., Dealers
76	Sawyers	Sawyers; Wood-Cutting Machinists
77	Wood Turners, Coopers, etc.	Wood Turners Wooden Box, Packing Case—Makers Coopers; Hoop—Makers, Benders Other Workers in Wood
78	Brick, Plain Tile, Terra Cotta—Makers	Brick, Plain Tile, Terra Cotta—Makers
79	Plaster, Cement—Manufacture	Plaster, Cement—Manufacture
80	Potters; Earthenware, etc.—Manufacture	Earthenware, China, Porcelain—Manufacture

TABLE 75—*Continued*

REFERENCE NUMBER	OCCUPATIONAL GROUPS, 1910–12	OCCUPATIONAL HEADINGS IN CENSUS REPORT
	Title	Title
81	Glass Manufacture	Sheet, Plate—Glass Manufacture
		Glass Bottle Manufacture
		Other Workers in Glass Manufacture
82	Chemical Manufacture	Manufacturing Chemists
		Alkali Manufacture
83	Chemists, Druggists	Chemists, Druggists
84	Tallow, Soap, Glue, Manure, etc.—Manufacture	Candle, Grease—Manufacture
		Soap—Boilers, Makers
		Manure Manufacture
		Glue, Size, Varnish, etc.—Makers
84*a*	Tallow, Soap, etc.—Manufacture	Candle, Grease—Manufacture
		Soap—Boilers, Makers
85	India Rubber, Gutta Percha—Workers; Waterproof Goods Makers	India Rubber, Gutta Percha—Workers
		Waterproof Goods Makers
86	Furriers, Skinners	Furriers, Skinners
87	Tanners	Tanners
88	Curriers; Leather Goods Makers	Curriers
		Leather Goods, Portmanteau, Bag, Strap, etc.—Makers
88*a*	Leather Goods, Portmanteau—Makers	Leather Goods, Portmanteau, Bag, Strap, etc.—Makers
89	Saddlers; Harness Makers	Saddlers; Harness, Whip—Makers
90	Brush, Broom—Makers; Hair, Bristle—Workers	Brush, Broom—Makers; Hair, Bristle—Workers
91	Paper Manufacture	Paper Manufacture
92	Stationery, Envelope, Cardboard Box, etc.—Manufacture	Stationery Manufacture
		Envelope Makers
		Paper Bag Makers
		Cardboard Box Makers
		Other Workers in Paper, etc.
92*a*	Stationery, etc.—Manufacture	Stationery Manufacture
		Envelope Makers
		Paper Bag Makers
		Other Workers in Paper, etc.
93	Dealers in Paper, Prints, Books and Stationery	Stationers, Law Stationers
		Other Dealers in Paper
		Book, Print—Publishers, Sellers
		Newspaper Agents, News Room Keepers
94	Printers	Printers—Hand Compositors
		Machine Compositors
		Printing Machine Minders
		Stereotypers, Electrotypers
		Others in Printing

TABLE 75—*Continued*

REFERENCE NUMBER	OCCUPATIONAL GROUPS, 1910–12	OCCUPATIONAL HEADINGS IN CENSUS REPORT
	Title	Title
94*a*	Printers—Hand Compositors	Printers—Hand Compositors
94*b*	Printers—Machine Compositors; Stereotypers, Electrotypers	Printers—Machine Compositors Stereotypers, Electrotypers
95	Lithographers; Copper and Steel Plate Printers	Lithographers; Copper and Steel Plate Printers
96	Bookbinders	Bookbinders
97	Cotton Manufacture	Cotton—Card and Blowing Room Processes Spinning Processes Winding, Warping, etc., Processes Weaving Processes Workers in other Processes Workers undefined
97*a*	Cotton—Strippers and Grinders	Cotton—Card and Blowing Room Processes (Strippers and Grinders)
97*b*	Blow Room Hands	Do. (Others)
98	Wool, Worsted—Manufacture	Wool—Sorting Processes Carding and Combing Processes Wool and Worsted—Spinning Processes Weaving Processes Workers in other Processes Workers, undefined
98*a*	Wool—Sorting, Carding and Combing Processes	Wool—Sorting Processes Carding and Combing Processes
99	Silk Manufacture	Silk—Spinning Processes Weaving Processes Workers in other Processes Workers undefined
100	Rope, Twine, Mat, Canvas, Sailcloth, etc.—Manufacture	Cocoa Fibre Manufacture Rope, Twine, Cord—Makers Mat Makers Canvas, Sailcloth, Sacking, Net, etc.—Manufacture.
101	Hosiery Manufacture	Hosiery Manufacture
102	Lace Manufacture	Lace Manufacture
103	Carpet, Rug, Felt—Manufacture	Carpet, Rug, Felt—Manufacture

TABLE 75—*Continued*

REFERENCE NUMBER	OCCUPATIONAL GROUPS, 1910–12	OCCUPATIONAL HEADINGS IN CENSUS REPORT
	Title	Title
104	Textile—Dyers, Bleachers, Printers, Finishers, etc.	Textile—Bleachers Printers Dyers Calenderers, Finishers, etc.
105	Drapers, Linen Drapers, Mercers	Drapers, Linen Drapers, Mercers
106	Straw Plait, Straw Hat, Straw Bonnet—Manufacture	Straw Plait Manufacture Straw Hat, Straw Bonnet—Manufacture
107	Hatters	Felt Hat Manufacture Makers of Cloth Hats and Caps Makers of Other Hats and Caps Hat, Bonnet, Straw Plait, etc.—Dealers
107*a*	Felt Hat Manufacture (Cheshire)	Felt Hat Manufacture
108	Tailors	Tailors Clothiers, Outfitters—Dealers
108*a*	Tailors (occupied only in certain boroughs where tailoring is largely carried on in factories)*	Tailors
108*b*	Tailors (in the boroughs of Stepney and Bethnal Green)	Tailors
109	Shoemakers	Boot, Shoe—Makers Slipper Makers Patten, Clog—Makers Boot, Shoe, Patten, Clog—Dealers
109*a*	Boot, Shoe, Slipper—Makers (Northamptonshire and Leicestershire)	Boot, Shoe—Makers Slipper Makers
109*b*	Patten, Clog—Makers (Lancashire)	Patten, Clog—Makers
110	Hairdressers	Wig Makers; Hairdressers
111	Milksellers, Dairymen	Milksellers, Dairymen
112	Provision Dealers	Cheesemongers, Buttermen, Provision Dealers
113	Butchers	Slaughterers Butchers, Meat Salesmen
114	Fishmongers, Poulterers	Fishmongers, Poulterers, Game Dealers
115	Millers; Cereal Food Manufacture	Millers; Cereal Food Manufacture
116	Corn, Flour, Seed—Merchants, Dealers	Corn, Flour, Seed—Merchants, Dealers
117	Bakers, Confectioners	Bread, Biscuit, Cake, etc.—Makers Bakers, Confectioners (Dealers)

* Bristol, Huddersfield, Norwich, Walsall, Wigan, and Plymouth.

TABLE 75—*Concluded*

REFERENCE NUMBER	OCCUPATIONAL GROUPS, 1910–12	OCCUPATIONAL HEADINGS IN CENSUS REPORT
	Title	Title
117*a*	Bread, Biscuit, Cake, etc.—Makers	Bread, Biscuit, Cake, etc.—Makers
118	Grocers	Grocers; Tea, Coffee, Chocolate—Dealers
119	Greengrocers, Fruiterers	Greengrocers, Fruiterers
120	Tobacco Manufacture	Tobacco Manufacture
121	Tobacconists	Tobacconists
122	Maltsters	Maltsters
123	Brewers	Brewers
124	Inn, Hotel—Keepers; Publicans; Spirit, Wine, Beer—Dealers.	Inn, Hotel—Keepers; Publicans, Beer sellers, Cider Dealers Wine and Spirit—Merchants, Agents
125	Inn, Hotel, etc.—Servants	Domestic Indoor Servants in Hotels, Lodging Houses and Eating Houses Barmen Waiters (not Domestic) Others in Inn, Hotel, Eating House, etc.—Service
125*a*	Barmen	Barmen
125*b*	Waiters	Waiters
126	Beer Bottlers, Cellarmen	Beer Bottlers Cellarmen
127	Gas Works Service	Gas Works Service
128	Electricity Supply	Electricity Supply
129	Chimney Sweeps	Chimney Sweeps
130	General Shopkeepers	Receiving Shop, Receiving Office—Keepers, Assistants (Laundry; Dyers and Cleaners) Multiple Shop, Multiple Store—Proprietors, Workers (general or undefined) General or Unclassified Shopkeepers; General Dealers
131	Costermongers, Hawkers	Costermongers, Hawkers, Street Sellers News—Boys, Vendors (street or undefined)
132	Engine—Drivers, Stokers, Firemen (not Railway, Marine, Agricultural or Electricity Supply)	Engine—Drivers, Stokers, Firemen (not Railway, Marine or Agricultural)

The table and instruction sheet were then given to each of the following persons, who made an independent classification of the occupations:

A. S. Dean, M.D.
J. R. Earp, L.R.C.P.
P. R. Hawley, M.D.
W. T. Howard, M.D.
A. J. Lotka, Ph.D.
Sylvia L. Parker, A.B.
Raymond Pearl, Ph.D.
L. J. Reed, Ph.D.
R. A. Spaeth, Ph.D.

All of these persons were either students in statistics or members of the staff of the School of Hygiene. One of the number (J. R. E.) is an Englishman, and another (A. J. L.) grew up as a boy in England. One (R. A. S.) is a specialist in industrial hygiene. No other instructions except those on the printed sheet were given.

The following occupations were decided by R. P. and P. R. H., after careful consideration and decision, to be *chiefly outdoor:* Reference numbers: 2, 12, 17, 19 to 43 inclusive, 59, 68, 69, 70, 72, 78, 79, 111, 131.

About this grouping there can be little question except on certain particular items. No. 17 (insurance agents) should perhaps be indoor; we made the decision on the basis of the habits of American insurance agents, who are outdoors more than they are indoors, taken as a class. Whether mining (nos. 36, 37, 38, 39) is to be regarded as an indoor or outdoor occupation is a difficult question. We finally thought the weight of evidence, all things considered, to be on the outdoor side. Carpenters and joiners (68) and painters and decorators (72) presented the difficulty that their work as a class is obviously partly indoor and partly outdoor. It seemed on the whole probable that more time, statistically considered, was spent in work out-of-doors than indoors in these occupations. Whether our decision is *right* in these doubtful cases it seems to us difficult to say with any precision. It is a matter of opinion mainly, and opinions will differ. However, little difference can possibly be made in the final result (as will presently appear) by the disposition of the few doubtful cases.

With the 9 independent classifications in hand the next step was to calculate, for each of the 132 occupations, the mean and standard deviation of position in the 5 group classification. Any occupation might theoretically be put in position I (lowest estimated energy expenditure), position II, III, IV or V. What was the *average* position (and its standard deviation) of each occupation on the basis of the 9 opinions taken?

The results in ascending order, are shown in table 76 for primarily indoor occupations. Since a number of occupations in different parts of the table showed identical mean positions the problem was presented of arranging these in such way as to carry out most consistently and logically the idea of an ascending order of estimated energy expenditure throughout the table. The following rules were adopted.

Where two or more occupations had the same mean position, that one was placed *lowest* which had a positive skewness of the positional distribution and the highest standard deviation. That one was placed *highest* which had a negative skewness of the positional distribution and the greatest standard deviation. In between these extremes the occupations with identical means were placed in order of descending magnitude of standard deviations with positive skewness, and ascending magnitudes with negative skewness. For example:

OCCUPATIONAL NUMBER	MEAN POSITION	SKEWNESS	STANDARD DEVIATION OF POSITION
124	2.333	+	0.667
105	2.333	+	0.471
112	2.333	−	0.667
118	2.333	−	0.667
119	2.333	−	0.667
13	2.333	−	0.943

Final differences in position, where no others obtained, were settled by merely the position of the reference number of the occupation—for example above 112, 118, 119.

This whole procedure was based on the theory that the *weight* of opinion among the 9 persons was towards one or the other extreme. For example:

Occupation 2. Actual distribution of position

	POSITION				
	I	II	III	IV	V
Frequency.....	1	2	5	1	0

That is one person placed this occupation in position I, 2 in position II, etc. Thus, while the distribution is negatively skew the weight of opinion is that this was a fairly average occupation, and the mean position was lowered by the three people who thought it less than average. Hence, all other things being equal, it was placed higher than had the opinions been distributed with positive skewness.

TABLE 76

Position of indoor occupations in ascending order of estimated expenditure of physical energy, as judged by nine persons independently

QUINTILE	REFERENCE NUMBER OF OCCUPATION	MEAN POSITION	DIRECTION OF SKEWNESS	STANDARD DEVIATION
I	3	1.000	0	0
	4	1.111	+	0.314
	15	1.444	+	0.685
	9	1.444	+	0.497
	10	1.444	+	0.497
	18	1.444	+	0.497
	16	1.556	+	0.685
	5	1.556	−	0.497
	7	1.556	−	0.497
	14	1.556	−	0.497
	8	1.667	+	0.816
	93	1.778	−	0.416
	1	1.778	−	0.628
	6	1.889	−	0.737
	44	2.000	0	0.471
	64	2.000	0	0.667
	110	2.000	0	0.667
	121	2.111	+	0.875
	75	2.111	+	0.567
II	130	2.222	+	0.628
	83	2.222	+	0.416
	124	2.333	+	0.667
	105	2.333	+	0.471
	112	2.333	−	0.667
	118	2.333	−	0.667
	119	2.333	−	0.667
	13	2.333	−	0.943
	120	2.444	+	0.497
	114	2.444	−	0.685
	63	2.556	−	0.497
	108	2.556	−	0.497
	116	2.556	−	0.497
	65	2.667	+	0.667
	101	2.667	+	0.667
	107	2.778	−	0.416
	102	2.778	−	0.628
	96	2.889	−	0.567
	106	2.889	−	0.567

TABLE 76—*Continued*

QUINTILE	REFERENCE NUMBER OF OCCUPATION	MEAN POSITION	DIRECTION OF SKEWNESS	STANDARD DEVIATION
	95	2.889	−	0.737
	113	2.889	−	0.994
	67	3.000	0	0.817
	90	3.000	0	0.471
	92	3.000	0	0.471
	94	3.000	0	0.471
	104	3.000	0	0.471
	122	3.000	0	0.817
	126	3.111	+	0.875
III	99	3.111	+	0.567
	128	3.111	+	0.567
	88	3.111	+	0.314
	103	3.222	+	0.786
	82	3.222	+	0.628
	84	3.222	+	0.628
	117	3.222	+	0.628
	98	3.222	+	0.416
	109	3.222	+	0.416
	47	3.333	+	0.817
	66	3.333	+	0.471
	86	3.333	+	0.471
	89	3.333	+	0.471
	91	3.333	+	0.471
	115	3.333	+	0.471
	123	3.333	+	0.471
	127	3.333	−	0.667
	11	3.333	−	0.816
	48	3.444	+	0.832
IV	60	3.444	+	0.832
	100	3.444	+	0.685
	85	3.444	+	0.497
	46	3.444	−	0.685
	125	3.444	−	0.685
	129	3.444	−	0.685
	74	3.667	+	0.667
	73	3.667	−	0.471
	76	3.667	−	0.471
	71	3.667	−	0.817
	77	3.778	+	0.916
V	80	3.778	+	0.416
	56	3.778	−	0.628

TABLE 76—*Concluded*

QUINTILE	REFFRENCE NUMBER OF OCCUPATION	MEAN POSITION	DIRECTION OF SKEWNESS	STANDARD DEVIATION
V	87	3.778	−	0.628
	97	3.778	−	0.628
	57	3.778	−	0.786
	52	3.889	−	0.567
	51	3.889	−	0.737
	62	4.111	+	0.567
	54	4.222	+	0.416
	55	4.222	+	0.416
	81	4.222	−	0.786
	58	4.333	+	0.667
	53	4.333	+	0.471
	61	4.333	+	0.471
	132	4.333	−	0.667
	45	4.778	−	0.416
	49	5.000	0	0
	50	5.000	0	0

TABLE 77

Distribution of positional standard deviations of table 76

VALUE OF STANDARD DEVIATIONS	FREQUENCY
0–0.19	3
0.20–0.39	2
0.40–0.59	43
0.60–0.79	34
0.80–0.99	13
Total.....	95

The theoretically possible values of the standard deviations of position range from 0 (when all 9 persons put an occupation in the same position), to 1.99 (when 5 put the occupation in one end position, and 4 put it in the other end position). The actual distribution of standard deviations of position for the indoor occupations of table 76 is that shown in table 77, and graphically in figure 50.

It is a difficult problem in probability, and one which we pass on to any pure mathematician interested in the subject, to determine what would be the distribution of 95 standard deviations if the placing of occupations by the 9 persons had been completely at random. We know, however, that the limits would be 0 and 1.99 approximately. Therefore in drawing figure 50 we have thought it desirable to put in, at least, the limits of the random distribution.

It is evident that the actual distribution covers only the lower half of the range of the theoretically possible distribution, on the basis of purely random assortment. The judgments of the 9 persons are, in short, in good agreement, or highly correlated, with each other.

As is indicated in table 76, after the 95 occupations were arranged in ascending order of estimated energy expenditure they were broken into five equal quintile groups. Quintile I, for example, includes the 19 indoor occupations which on the averaged judgment of the 9 persons, involved the lowest expenditure of physical energy in their pursuit. Quintile II contains the 19 occupations on the average judged to involve a larger expenditure of energy than those in I, and a smaller expenditure than those in III, and

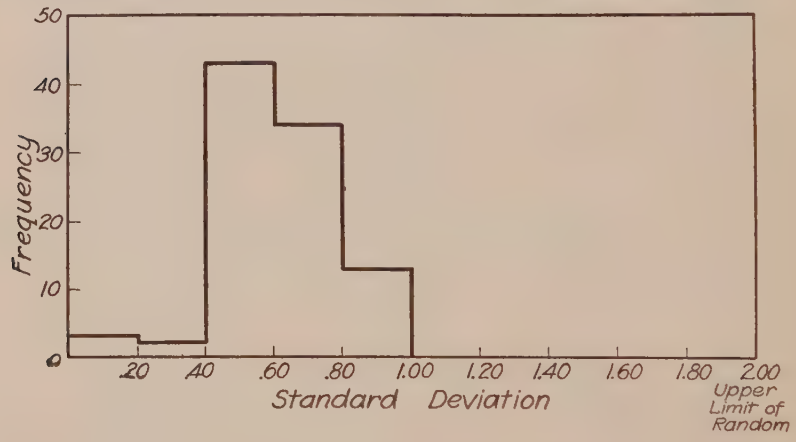

FIG. 50. FREQUENCY DISTRIBUTION OF THE OBSERVED POSITIONAL STANDARD DEVIATIONS FOR THE 95 CHIEFLY INDOOR OCCUPATIONS

so on. It is with these broad quintile groups that we shall work from now on in our discussion of mortality.

The corresponding data for the *chiefly outdoor* occupations are given in table 78. This is arranged on precisely the same plan as table 76, and the figures and arrangement were arrived at in the same way.

The distribution of the standard deviations of table 78 is given in table 79 and graphically in figure 51.

The distribution is fundamentally like that for the indoor occupations, but indicates much less unanimity of judgment in the placing of the outdoor occupations than was displayed in the case of the indoor. Perhaps this is a reflection of the fact that the nine persons who did the estimating are themselves "chiefly indoor" workers, and have as a group less knowledge

TABLE 78

Position of outdoor occupations in ascending order of estimated expenditure of physical energy, as judged by nine persons independently

QUINTILE	REFERENCE NUMBER OF OCCUPATION	MEAN POSITION	DIRECTION OF SKEWNESS	STANDARD DEVIATION
I	17	1.444	+	0.685
	24	2.000	+	0.943
	23	2.000	+	0.817
	26	2.111	+	0.994
	31	2.111	+	0.737
	20	2.444	+	0.832
	131	2.556	+	0.956
II	111	2.667	+	0.943
	2	2.667	−	0.817
	72	2.778	−	0.628
	25	3.000	+	0.943
	12	3.000	0	0.817
	34	3.222	+	0.628
	41	3.222	+	0.628
	68	3.333	+	0.667
III	27	3.444	+	0.832
	40	3.444	−	0.956
	70	3.556	+	0.685
	32	3.556	−	0.832
	35	3.667	+	1.054
	69	3.667	+	0.667
	78	3.667	−	0.471
	79	3.667	−	0.471
IV	59	3.778	−	0.628
	21	3.889	−	0.737
	19	4.000	0	0.667
	28	4.111	+	0.737
	22	4.222	+	0.416
	33	4.333	+	0.471
	42	4.444	+	0.497
V	43	4.556	−	0.497
	29	4.667	−	0.471
	38	4.667	−	0.471
	39	4.667	−	0.471
	37	4.778	−	0.416
	36	4.889	−	0.314
	30	5.000	0	0

and experience of what different sorts of outdoor labor really mean. Furthermore, there was a great unfamiliarity with both the duties and the designations of certain *English* outdoor occupations.

In setting the quintile limits in table 78 it will be noted that two slight liberties have been taken with the data. Quintiles II and III each contain 8 occupations, while each of the other three contain 7. This was done to avoid pro-rating the death rates of certain occupations between adjacent

TABLE 79

Distribution of positional standard deviations of table 78

VALUE OF STANDARD DEVIATION	FREQUENCY
0–0.19	1
0.20–0.39	1
0.40–0.59	10
0.60–0.79	12
0.80–0.99	12
1.00–1.19	1
Total	37

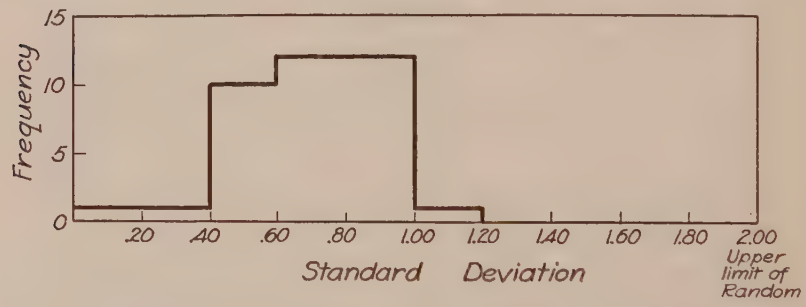

FIG. 51. FREQUENCY DISTRIBUTION OF THE OBSERVED POSITIONAL STANDARD DEVIATIONS FOR THE 37 CHIEFLY OUTDOOR OCCUPATIONS

quintiles, which would have been necessary had the quintile limits been placed exactly to make equal areas.

Because of the lack of first-hand knowledge, on the part of a majority of the 9 persons who placed the occupations, of the nature of the actual work incident to certain British outdoor occupations, and of the meaning of the English designations of these occupations, a few of the outdoor occupations are obviously and palpably in the wrong places. After due consideration of the matter, and the reflection that the fundamental purpose of this

study is *not* to examine the errors of judgment of a group of statisticians, but rather to find out if possible what the relation is between severity of physical labor and mortality, we have ventured to shift the positions of 6 of the 37 outdoor occupations from those in which they are found in table 78. We believe that by these changes a much fairer representation of the true facts is attained.

The occupations so changed, and the reasons are as follows: Occupation 20, Railway guards, porters, pointsmen, etc., is shifted from quintile I to quintile II. While it is true that the duties of a railway guard are not very strenuous physically, those of a railway porter are quite decidedly so. We believe that this group, as a group, comes on the average nearer to the second quintile of chiefly outdoor occupations than to the first quintile.

Occupation 31, Messengers, porters, watchmen (not railway or government), is shifted from quintile I to quintile II. We believe that this class was wrongly placed by most of our associates, because their minds were filled with the idea of the behavior of American messengers and watchmen. Our own observation leads strongly to the view that English messengers and watchmen do a great deal more hard work than do their American colleagues employed in the pursuits which go by the same names. And finally, there can be no doubt that the work of porters does not belong in the class of *easiest* occupations chiefly outdoor.

The worst mistake of placing of all is that of no. 131, costermongers, hawkers. A majority of our American associates plainly had no just conception of the life that the English costermonger or hawker lives. A bearer of often heavy burdens, or pusher of heavy carts, practically on his feet throughout the working day, he is distinctly libelled by being put in the easiest fifth of outdoor occupations. After careful consideration we have shifted this occupation from quintile I to quintile IV.

To replace these occupations moved from quintile I, three other occupations, originally wrongly placed (in our judgment) in quintile II, are put in quintile I. The first of these is occupation 2, civil service messengers. This is a mixed group. Some are strictly indoor workers, but it was thought that a majority of the working time of the whole group was spent out of doors. But taken as a group, there appears no warrant for placing it higher than quintile I in the scale of energy expenditure.

Occupation 12, gamekeepers, is shifted back to quintile I. There are few chiefly outdoor occupations *less* strenuous than that of gamekeeper. The case needs no argument. Our American colleagues perhaps confused an English gamekeeper with an American guide. There is a vast difference in reality.

Occupation 34, gardeners, nurserymen, seedsmen, is the third change from quintile II of the original grouping to quintile I. This is one of the most difficult of all the occupations to place correctly in our energy expenditure scheme. On the whole, however, it seems to us that it rightly belongs in the lowest fifth of outdoor occupations. It rarely involves really heavy work, but rather represents on the average what may be called the "more or less busy puttering around" type of job.

With these shifts the constitution of the quintiles for outdoor occupations becomes as follows:

Revised distribution of chiefly outdoor occupations

QUINTILE I	QUINTILE II	QUINTILE III	QUINTILE IV	QUINTILE V
2	20	27	19	29
12	25	32	21	30
17	31	35	22	36
23	41	40	28	37
24	68	69	33	38
26	72	70	42	39
34	111	78	59	43
		79	131	

We believe this to be an arrangement much more closely in accord with the true facts than that of table 78, and we shall accordingly use it in the discussion of the mortality of outdoor occupations in a later section.

It will have been noted that on the basis of table 78 there is a distinct difference in the positional placing of indoor and outdoor occupations. Averaging mean positions for the 95 and 37 occupations we have:

Mean of positional means, 95 indoor occupations = 2.97
Mean of positional means, 37 outdoor occupations = 3.49

In other words, the average position of outdoor occupations in the fivefold scale of estimated energy expenditure came out roughly one-half of a positional class *higher* than the average of the indoor occupations. Under the instructions laid down, if the placing of indoor and outdoor occupations had been done quite independently and without reference to each other, these means should have been the same, because each should represent the mid-point of a substantially normal distribution. Actually the estimating of position of the indoor and outdoor occupations was probably done by nearly every person at the same time. There thus arose a bias, chiefly unconscious but in some cases certainly conscious, from an attempt to

calibrate the outdoor scale against the indoor on a basis of absolute energy expenditure. From the outstart it was not our intention that there should be any such comparison or calibration. It is unfortunate that we did not foresee this outcome and by the organization of the judging compel an entirely independent placing of indoor and outdoor occupations. It must be said, however, that we are unable to see that the situation described in any way vitiates, or indeed sensibly influences, the results regarding mortality.

The age specific death rates for the 132 occupational groups are given in table 80. It is to be understood, in accordance with the argument developed earlier in this chapter, that in calculating these rates, we have in every case deducted the deaths (if any) recorded as due to accident. We have further deducted the deaths (if any) noted in the original Report as due to specific hazards of the occupation itself. What these latter deductions were is stated in each case, where any were made, in the last column of table 80, headed Remarks.

We now have in hand the data necessary for the discussion of our problem, to which we may at once proceed.

MORTALITY RESULTS. INDOOR OCCUPATIONS

In table 81 are presented the net death rates, after the deductions explained above, by quintiles and ages, for the "chiefly indoor" occupations. These death rates are *weighted* mean rates, the weighting being on the basis of the number of persons engaged in each occupation in the quintile. Thus, to get the figures in table 81, the populations (times 3) engaged in each occupation in a particular quintile, say I, were added together. The sum was the denominator of the rate fraction for that quintile (and age group), of which the numerator was the similarly summed deaths of three years after deductions. The probable errors were computed from the formula

$$\text{P.E.} = \pm 674.5 \sqrt{\frac{p\,q}{n}}$$

The actual populations and deaths from which the rates of table 81 are computed, are given in table 82.

The question now to be discussed is this: Is the mortality significantly higher in the higher quintiles than in the lower? And, in general, does the mortality rate increase as the energy expenditure increases, as indicated in the broad quintile grouping of occupations?

TABLE 80

Specific death rates from all causes per 1000 living in each corresponding group of males, by age and occupation, computed from data of original Report, with deaths due to accident deducted in all cases, and deaths from occupational hazards deducted where noted

REFERENCE NUMBER OF OCCUPATION	AGE GROUPS								REMARKS
	15–19	20–24	25–34	35–44	45–54	55–64	*Caution:* see p. 303 *supra* 65–74	75 and over	
1	1.52	2.17	3.33	5.88	10.07	21.62	51.20	125.00	
2	1.37	3.35	3.23	5.64	10.80	24.23	70.53	212.62	
3		1.33	2.08	3.01	7.44	21.38	50.07	136.75	
4		1.55	2.72	5.47	11.82	25.38	50.76	148.31	
5	1.49	2.88	4.71	7.49	13.94	27.28	64.71	160.83	
6		4.27	3.33	6.55	13.36	25.71	56.88	145.68	
7	1.54	3.20	2.50	3.92	8.70	22.66	55.22	138.28	
8	2.22	3.33	3.63	6.78	13.19	25.16	70.97	180.61	
9	2.60	5.56	5.43	7.04	12.93	32.11	62.45	187.18	
10	2.37	3.52	6.55	10.33	17.46	30.83	66.41	159.52	
11	1.82	2.53	3.88	7.42	11.66	31.00	61.57	195.81	
12	1.61	1.69	1.39	2.88	8.59	23.04	70.35	176.40	
13	1.97	2.27	3.23	6.08	12.81	32.01	68.88	205.39	
14	1.98	3.77	4.98	8.45	16.04	31.50	68.21	167.77	
15	1.25	1.71	2.24	3.48	10.02	24.34	52.83	131.23	
16	1.37	2.36	2.38	3.42	10.26	22.76	59.06	105.00	
17	2.31	3.27	5.26	7.31	12.56	26.91	48.09	132.48	
18	1.67	2.67	3.90	4.75	10.16	24.34	64.76	187.32	
19	2.17	2.52	2.38	3.55	8.33	24.12	61.67	191.04	
20	1.70	2.62	3.00	4.83	9.54	21.96	51.83	129.21	
21	2.31	1.81	2.73	4.21	8.65	21.77	70.64	232.80	
22	4.21	4.71	5.15	11.05	15.45	31.55	87.84	271.45	
23	1.37	2.88	4.51	8.99	17.09	33.24	71.35	200.11	
24	1.11	1.86	2.28	4.32	7.36	10.88	28.57	66.67	
25	1.90	3.08	4.52	8.41	14.95	32.06	80.85	233.08	1 death from occupational lead poisoning deducted
26	1.79	2.41	3.83	5.33	9.89	25.55	65.66	152.38	
27	3.76	6.52	9.72	13.52	23.94	41.95	82.16	169.18	1 death from occupational lead poisoning deducted
28	2.04	3.74	4.42	9.01	17.67	36.32	74.49	219.99	
29	1.38	3.63	6.59	11.16	20.27	34.19	64.67	163.56	2 deaths from anthrax deducted
30	1.49	2.39	4.53	9.44	17.88	31.89	69.13	217.46	
31	1.49	5.26	6.45	12.13	20.63	38.37	70.97	188.48	

TABLE 80—*Continued*

REFERENCE NUMBER OF OCCUPATION	AGE GROUPS								REMARKS
	15–19	20–24	25–34	35–44	45–54	55–64	*Caution:* see p. 303 *supra* 65–74	75 and over	
32	0.50	1.30	2.91	4.22	8.08	19.27	50.29	158.16	1 death from anthrax deducted
33	1.21	2.23	2.85	4.48	7.51	16.46	44.48	168.90	1 death from occupational lead poisoning and 4 from anthrax deducted
34	1.13	2.02	2.90	4.09	7.69	17.67	43.36	148.26	
35	1.61	3.37	5.39	9.08	13.36	24.74	58.07	173.80	
36	1.85	2.53	3.01	5.13	10.79	27.77	79.98	218.89	3169 deaths due to mine accidents (in addition to regular rubric "accidents") deducted
37	1.01	2.74	2.77	5.18	8.90	26.00	63.70	207.07	
38	1.42	5.52	6.83	20.10	30.95	45.80	106.06	255.71	
39		1.04	5.63	8.32	20.00	49.66	132.25	260.42	
40	2.06	3.12	2.44	3.01	9.87	20.16	75.16	250.00	
41	3.47	3.75	4.27	9.19	15.98	40.40	50.00	222.22	
42	1.83	2.45	4.19	8.13	17.21	34.59	71.16	200.17	115 deaths from fibroid disease of lungs and 1 from occupational lead poisoning deducted
43	3.73	4.86	4.69	7.13	13.85	36.61	80.03	166.67	4 deaths from fibroid disease of lungs deducted
44	0.30	2.97	2.79	5.29	10.79	24.20	58.98	201.43	
45	1.99	3.31	3.91	6.69	12.97	28.99	74.48	182.14	6 deaths from occupational lead poisoning deducted
46	1.53	2.51	3.20	5.17	10.04	20.69	49.48	152.48	3 deaths from occupational lead poisoning deducted
47	1.54	3.21	4.62	10.25	19.70	35.86	82.69	202.04	27 deaths from fibroid disease of lungs and 3 from occupational lead poisoning deducted
48		4.23	6.16	10.71	15.78	35.81	79.02	195.12	1 death from occupational lead poisoning deducted
49	1.84	3.01	4.16	7.40	15.08	32.76	77.81	225.06	3 deaths from occupational lead poisoning deducted
50	1.67	2.88	3.69	6.22	14.19	30.29	71.90	186.12	
51	2.01	2.24	3.56	8.74	18.05	30.72	71.05	273.02	
52	2.19	2.98	3.91	5.57	12.66	27.16	70.00	233.33	
53	1.32	2.42	3.49	6.38	12.33	24.19	70.96	164.95	

TABLE 80—*Continued*

REFERENCE NUMBER OF OCCUPATION	AGE GROUPS								REMARKS
	15–19	20–24	25–34	35–44	45–54	55–64	*Caution:* see p. 303 *supra* 65–74	75 and over	
54			0.82	3.53	12.42	71.79	64.10	55.56	3 deaths from occupational lead poisoning deducted
55	2.56	4.17	3.44	7.93	14.73	31.70	74.23	221.01	2 deaths from occupational lead poisoning deducted
56	2.17	3.80	7.22	10.63	18.78	37.69	72.12	191.41	1 death from occupational lead poisoning deducted
57	1.47	4.53	4.95	8.10	15.29	30.98	111.11	222.22	11 deaths from occupational lead poisoning deducted
58	2.12	4.27	5.78	9.12	15.80	36.86	86.77	197.41	1 death from occupational lead poisoning deducted
59	1.71	2.64	3.16	5.81	9.92	22.99	59.49	160.96	3 deaths from occupational lead poisoning deducted
60	1.49	2.87	4.35	7.24	13.86	20.54	57.42	145.83	
61	1.08	1.78	2.18	4.36	9.23	21.38	58.60	173.89	13 deaths from occupational lead poisoning deducted
62	1.73	1.90	2.65	6.15	10.39	27.10	67.09	170.48	1 death from occupational lead poisoning deducted
63	2.07	2.70	2.85	5.46	10.12	24.61	63.08	164.31	
64	1.94	3.32	5.06	7.11	13.37	31.06	71.85	182.52	1 death from occupational lead poisoning deducted
65	1.88	1.84	4.31	4.95	10.40	27.45	57.30	196.43	2 deaths from occupational lead poisoning deducted
66	1.50	4.25	4.54	7.25	12.64	26.46	60.08	151.80	
67	0.78	2.04	2.54	4.34	11.76	29.99	75.77	193.28	1 death from occupational lead poisoning deducted
68	1.84	2.99	3.55	6.10	12.50	26.90	63.24	161.51	
69	1.76	2.07	3.17	5.20	12.27	24.17	55.91	157.31	
70		0.90	4.93	8.05	19.02	33.56	70.89	178.03	
71	2.05	2.30	3.51	6.20	15.14	28.17	65.93	169.56	
72	1.47	2.86	3.94	7.80	15.55	32.05	73.37	181.44	140 deaths from occupational lead poisoning deducted
73	1.61	2.88	3.19	6.65	14.03	32.22	72.15	178.85	
74	1.91	3.70	4.74	7.47	14.74	28.61	64.23	188.51	
75	1.32	2.17	2.59	4.85	10.29	19.78	60.34	135.46	
76	1.20	2.67	2.32	4.85	8.89	22.02	67.29	194.73	
77	2.43	3.85	4.25	7.46	14.10	31.52	65.78	178.17	2 deaths from occupational lead poisoning and 6 from asthma deducted

TABLE 80—*Continued*

REFERENCE NUMBER OF OCCUPATION	AGE GROUPS								REMARKS
	15–19	20–24	25–34	35–44	45–54	55–64	*Caution:* see p. 303 *supra* 65–74	75 and over	
78	1.42	3.17	3.22	4.93	9.04	22.71	62.42	208.14	
79	1.30	3.23	2.48	4.83	8.62	21.54	58.77	233.83	2 deaths from asthma and 6 from fibroid disease of lungs deducted
80	2.44	3.25	5.22	9.90	23.05	45.07	90.96	218.50	25 deaths from occupational lead poisoning; 22 from asthma, and 28 from fibroid disease of lungs deducted
81	2.12	4.74	5.02	8.26	18.63	33.68	87.23	232.07	1 death from occupational lead poisoning deducted
82	1.95	2.61	2.57	5.10	11.34	28.38	60.81	156.21	
83	2.67	3.07	4.54	7.14	11.92	28.81	65.06	156.94	
84	2.03	3.48	1.86	4.37	7.90	19.58	47.53	194.23	
85	2.08	3.59	4.50	7.29	11.65	28.50	63.44	222.22	
86	2.06	2.84	3.49	8.68	18.50	31.51	79.56	236.84	
87	1.58	2.41	3.87	4.85	10.99	24.74	65.52	236.02	1 death from anthrax deducted
88	2.01	3.47	5.37	7.91	14.46	27.90	73.12	179.22	1 death from anthrax deducted
89	0.73	3.76	3.18	7.09	13.68	29.73	66.17	204.88	
90	1.55	4.37	5.31	9.13	17.70	30.50	57.61	207.10	
91	2.45	2.12	3.80	4.33	11.97	29.57	65.85	210.88	
92	2.61	4.66	4.34	5.32	12.96	24.92	42.15	158.12	
93	2.11	4.39	4.62	6.65	10.52	23.91	54.22	164.91	
94	2.13	4.28	4.59	7.61	14.04	27.84	64.84	162.37	7 deaths from occupational lead poisoning deducted
95	1.06	3.52	2.54	4.76	10.84	23.46	62.89	150.54	
96	2.03	4.10	5.80	7.02	15.09	25.96	55.86	141.55	
97	2.55	3.49	3.62	6.25	14.66	35.83	97.97	256.66	
98	2.57	3.57	4.32	6.79	13.94	33.75	86.23	254.50	11 deaths from anthrax and 1 from occupational lead poisoning deducted
99	3.07	2.69	2.71	6.65	14.47	29.61	75.03	223.23	
100	1.53	5.10	3.57	5.00	11.51	27.54	58.02	198.90	4 deaths from asthma deducted
101	1.94	3.89	5.28	6.52	11.88	28.97	67.52	206.32	
102	3.03	3.40	4.35	8.83	11.95	30.28	74.66	232.80	
103	3.24	5.07	3.63	3.90	11.70	28.63	86.11	189.45	

TABLE 80—*Concluded*

REFERENCE NUMBER OF OCCUPATION	AGE GROUPS								REMARKS
	15–19	20–24	25–34	35–44	45–54	55–64	*Caution:* see p. 303 *supra* 65–74	75 and over	
104	2.51	3.73	4.43	7.27	15.80	34.54	89.55	249.16	1 death from occupational lead poisoning deducted
105	1.96	2.60	3.50	5.96	11.07	25.74	60.85	162.68	
106	0.47	3.97	3.13	7.24	14.01	26.07	57.66	219.70	
107	1.70	3.04	3.87	7.32	16.76	26.13	67.63	179.92	
108	1.71	3.43	4.34	7.65	14.86	30.43	68.49	169.44	
109	2.71	4.44	5.27	8.27	14.58	29.50	66.51	182.36	
110	2.46	3.57	5.42	9.14	17.68	34.22	65.48	162.96	
111	1.62	3.38	4.34	6.81	13.47	27.38	74.26	227.12	
112	1.09	2.19	3.43	5.85	13.39	29.23	64.23	200.00	
113	1.27	2.56	3.99	7.82	17.51	35.18	67.52	169.82	3 deaths from anthrax deducted
114	1.59	2.63	3.90	7.55	14.02	27.72	63.10	198.23	
115	1.50	2.55	2.52	4.81	10.59	29.48	63.80	209.21	
116	1.19	1.49	3.56	4.78	9.02	22.73	62.86	169.37	
117	1.74	2.67	3.26	5.90	12.41	26.53	59.02	169.75	18 deaths from asthma deducted
118	1.52	2.63	3.35	5.77	11.08	25.46	61.01	164.09	
119	1.45	3.78	4.38	8.72	12.27	26.30	72.47	225.47	
120	1.34	2.70	4.42	5.46	13.48	30.78	66.45	170.07	
121	1.50	4.85	6.85	8.49	12.78	24.67	60.00	139.64	
122	1.91	0.85	1.91	5.01	8.63	27.59	66.79	223.74	
123	3.29	3.05	6.55	9.40	19.63	35.50	72.49	180.81	
124	1.19	2.08	7.72	13.77	23.95	40.94	84.54	216.20	
125	1.80	3.26	7.94	14.04	22.33	33.33	58.70	145.73	
126	1.53	3.92	5.97	9.13	16.79	36.69	76.04	153.01	
127	1.52	2.72	3.56	6.36	12.69	28.05	70.08	211.74	
128	0.87	1.52	1.62	3.78	8.62	17.68	29.13	119.05	1 death from occupational lead poisoning deducted
129	4.17	3.20	4.28	12.02	18.42	35.44	64.33	198.88	
130	1.94	2.72	5.51	8.19	15.98	25.50	55.88	157.63	
131	3.14	6.30	10.83	17.75	26.73	43.10	73.57	221.88	
132	2.06	2.10	3.01	5.36	10.81	29.39	73.74	206.74	

TABLE 81

Weighted mean quintile death rates per 1000 at ages, for indoor occupations

QUINTILE	AGE GROUPS							
	15–19	20–24	25–34	35–44	45–54	55–64	65–74 (see p. 303)	75 and over (see p. 303)
I	1.86±0.04	3.31±0.06	4.12±0.05	6.43±0.07	12.19±0.11	26.06±0.21	59.85±0.44	153.54±1.25
II	1.73±0.05	2.84±0.06	4.18±0.05	7.73±0.08	14.76±0.13	30.04±0.23	67.65±0.48	181.28±1.32
III	2.05±0.05	3.44±0.06	4.12±0.05	7.10±0.07	14.22±0.12	30.31±0.23	69.94±0.47	189.12±1.30
IV	1.72±0.05	3.00±0.07	4.33±0.06	7.33±0.09	14.07±0.15	29.21±0.28	66.06±0.61	190.35±1.92
V	2.01±0.04	3.08±0.05	3.80±0.04	6.68±0.06	13.75±0.10	30.90±0.19	75.94±0.42	200.93±1.35

TABLE 82

Absolute figures for populations and deaths, indoor occupations

QUINTILE	AGE GROUPS															
	15–19		20–24		25–34		35–44		45–54		55–64		65–74		75 and over	
	*A**	*B**	*A*	*B*	*A*	*B*	*A*	*B*	*A*	*B*	*A*	*B*	*A*	*B*	*A*	*B*
I	473,373	882	463,482	1,535	791,340	3,262	606,306	3,899	432,000	5,267	250,830	6,536	122,577	7,336	37,749	5,796
II	335,298	579	314,652	895	628,695	2,625	579,030	4,477	410,844	6,065	255,147	7,665	126,756	8,575	38,967	7,064
III	446,136	914	380,874	1,312	679,404	2,798	586,554	4,164	409,488	5,824	253,437	7,682	135,171	9,454	41,255	7,804
IV	295,263	508	305,547	917	563,298	2,440	408,144	3,011	274,395	3,860	159,102	4,648	74,067	4,893	18,897	3,597
V	705,576	1,420	650,406	2,004	1,183,530	4,502	956,286	6,391	658,386	9,056	392,160	12,118	175,566	13,333	40,203	8,078

* In this table *A* denotes population at 1911 census multiplied by 3, because deaths were taken for 3 years; *B* denotes deaths for the three years 1910, 1911 and 1912.

From table 81 we note the following points:

1. The slope or trend of the death rates is generally *upward* from quintile I to V. The magnitude of the slope is, however, small in some of the age groups; so small in point of fact, as to be insignificant.

2. In the early age groups (up to 45 years of age) the trends of the death rates are generally small in magnitude, and from 45 on larger. The explanation which obviously suggests itself is that in the earlier half of life the specific forces of mortality in general are so low (or put the other way about, the natural, inborn healthfulness or power of resistance of the organism is so high) that it makes no great difference to a group of men in respect of their mortality rate, *within those earlier ages*, whether they work at hard or easy tasks. In short, it is difficult to kill a man by physically hard work before he is 40 years of age, occupational accidents and hazards being always excluded. After age 45 is past the case is progressively altered. The phenomena which are generically classified as those of senescence are definitely and perceptibly coming into operation. Organic changes in a retrograde direction are occurring. Whether a man is then, or in the prior years of his life has been, doing heavy or light physical labor may make a marked difference in his survival chances.

3. From age 35 on the most marked upward trend is from quintile I to II. There is little slope from quintile II to IV, and again a tendency towards an upward slope from IV to V. This may be taken to indicate what all of the 9 persons who classified the occupations felt in making the classification. It was comparatively easy to make a group of physically *very easy* occupations, and to make a group of physically *very hard* occupations. But in between these extremes one did not feel particularly sure of gradations.

This consideration leads to another way of testing our conclusion. Any fair-minded person will be bound to admit, we think, if he will take the trouble to read over table 76, in connection with table 75, that the occupations which form quintile V demand, and in actual practice entail, an expenditure of physical energy far in excess of the expenditure involved in the pursuit of the occupations in quintile I. Suppose we examine the differences in the death rates, with their probable errors, between these two extreme groups, for each age class, calling a difference + when the rate for quintile V is in excess of that for quintile I. The results are shown in table 83.

This table confirms in the most striking way our conclusions. Up to age 45 the differences between quintiles I and V in mortality are insignificant or negative. From age 45 on the differences become increasingly larger. In the 45 to 54 group the difference is 10 times the probable error and in

the 55 to 64 group 17 times. The increases in mortality are 13 and 19 per cent in these two groups.

The difference column of table 83 is shown graphically in figure 52.

TABLE 83

Differences in mortality rates between Quintile I and Quintile V. Indoor occupations

AGE GROUPS	DIFFERENCE V — I	RATIO OF DIFFERENCE TO ITS PROBABLE ERROR	PERCENTAGE INCREASE, (OR DECREASE) IN MORTALITY RATE IN PASSING FROM QUINTILE I TO V
			per cent
15–19	+0.15±0.06	2.7	+8.1
20–24	−0.23±0.08	2.9	−6.9
25–34	−0.32±0.06	5.0	−7.8
35–44	+0.25±0.09	2.7	+3.9
45–54	+1.56±0.15	10.5	+12.8
55–64	+4.84±0.28	17.1	+18.6
65–74*	+16.09±0.61	26.5	+26.9
75 and over	+47.39±1.84	25.8	+30.9

* See page 303 regarding this and the next age group.

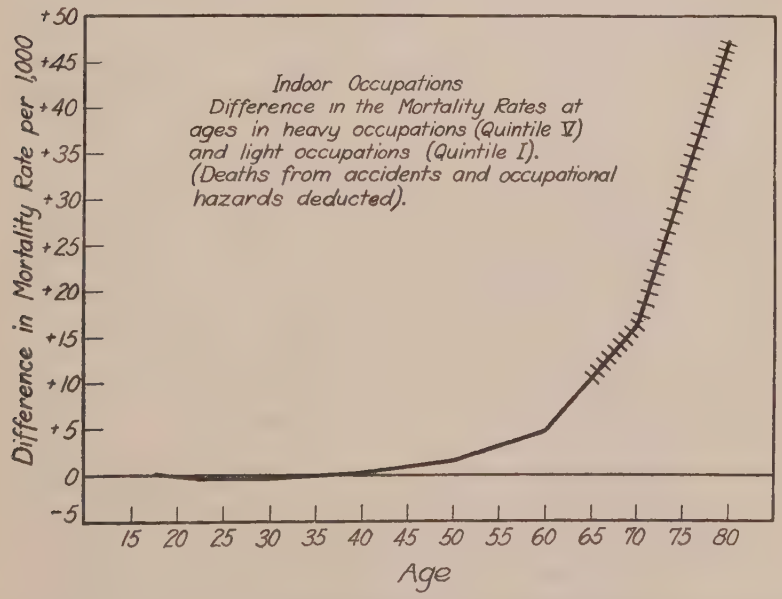

FIG. 52. DIFFERENCES IN THE MORTALITY RATES AT AGES BETWEEN HEAVY OCCUPATIONS (QUINTILE V) AND LIGHT OCCUPATIONS (QUINTILE I). INDOOR OCCUPATIONS

The line is crossed from age 65 on to indicate the uncertainty as to its true position at advanced ages.

So far we have been considering groups of *persons*. The weighted mean mortalities of table 81 are the death rates of large groups of persons, taken as population groups, in 5 lots graded according to estimated energy expenditure in the business of their lives. It is possible to look at the matter in another way. Suppose we regard the death rate attached to each of the 132 occupations of table 75 as an *attribute of that occupation*. Then the unweighted mean of the death rates proper to each of the 19 occupations forming quintile I will represent what the death rate of that quintile group would be if an equal number of persons engaged in each of the occupations in it. The only underlying assumption in this procedure which needs discussion is that in each of the ultimate occupation groups of table 75 there are engaged enough persons at each age to give a stable, fair, characteristic rate, which may truly be regarded as an *attribute* of the group. Actually this requirement is satisfied in all but a very few of the 1056 elemental groups involved in the discussion.

Why is it desirable to discuss the matter at all in this way? For the simple reason that a weighted mean quintile mortality may be extremely misleading if it happens that one *occupation* in it involves a large proportion of all the *persons* in the quintile group, and the mortality in that occupation is peculiarly distributed. Let an example clarify the point. Suppose, for simplicity's sake, that there were 10 occupations in each quintile instead of 19. And further suppose that the facts were as follows, at a certain age, say 15 to 19, see table on following page.

The essential characteristics of this arrangement are seen to be these. There is one occupation (A) in each quintile (of course a *different* occupation in each one) which involves a number of persons 10 times as large as the number involved in any other occupation. It furthermore is supposed to be the case that this largely followed occupation in quintile I has a relatively high death rate, whereas the corresponding occupation (*i.e.*, the one involving many people) in quintile V has a low death rate. All the other occupations in all the quintiles show the relation of increasing death rates in passing from I to V.

Now the rates given in the "total" line of the table are the weighted mean rates corresponding to those of table 81 above. These weighted means show a nearly horizontal but slightly *declining* trend from quintile I to V. But this result is contrary to the trend of 45 out of the 50 occupations involved in the case. Five occupations, with peculiar death rates, *and* a large population involved, swing the trend to the opposite of that characteristic of the vast majority of the *occupations*. The true state of the case, so far as *occupations* are concerned, is shown by the last line of unweighted mean rates, which give an upward trend from I to V.

LINE	QUINTILE I			QUINTILE II			QUINTILE III			QUINTILE IV			QUINTILE V		
	Population	Deaths	Rate	Population	Deaths	Rate	Population	Deaths	Rate	Population	Deaths	Rate	Population	Deaths	Rate
A	100,000	500	5	100,000	400	4	100,000	300	3	100,000	200	2	100,000	100	1
B	10,000	10	1	10,000	20	2	10,000	30	3	10,000	40	4	10,000	50	5
C, D, E, F, G, H, I, J	All these exactly like B														
Totals	190,000	590	3.1	190,000	580	3.05	190,000	570	3.0	190,000	560	2.9	190,000	550	2.89
Unweighted mean rates.			1.4			2.2			3.0			3.8			4.6

Now in certain cases something like this hypothetical illustration is actually found in our present statistics. For example, in the age group 15 to 19, of the total population ($\times$ 3) exposed to risk in quintile I of 473,373, more than half (263,502) are in one occupation, viz., 14, commercial clerks. Similar situations prevail in other quintiles. Consequently it is desirable to examine the unweighted mean death rates. These are given in table 84.

Comparison of the unweighted with the weighted mean mortality rates shows at once that it makes no essential difference in the results whether the problem is considered on the basis of occupations or persons. The unweighted mean trends are perhaps a little smoother, but on the whole must be regarded simply as confirming, from another point of view, the results already reached.

TABLE 84

Unweighted mean quintile death rates per 1000 at ages, for indoor occupations

QUINTILE	AGE GROUPS							
	15–19	20–24	24–34	35–44	45–54	55–64	65–74 (see p. 303)	75 and over (see p. 303)
I	1.63	3.14	3.95	6.22	12.15	25.94	60.23	155.81
II	1.72	2.87	4.29	7.07	13.37	28.16	65.13	186.13
III	1.95	3.30	3.73	6.49	13.43	28.85	66.81	186.17
IV	1.80	3.16	4.17	7.47	14.09	29.17	65.24	189.09
V	1.86	3.01	3.93	6.99	14.43	33.25	76.39	201.30

So then the general conclusion reached from the study of the indoor occupations is that there is a tendency for higher rates of mortality to be associated with occupations requiring larger expenditures of physical energy in their pursuit. This tendency is slight in the earlier age groups, but becomes very distinct after middle life is reached.

MORTALITY RESULTS. OUTDOOR OCCUPATIONS

Proceeding in exactly the same way with outdoor occupations that we have in the preceding section with indoor, we get table 85, the quintile distributions being those set forth on page 324 *supra*.

The actual populations and deaths from which the rates of table 85 are computed, are given in table 86.

The trends of the death rates are rather irregular, especially in the earlier years. The death rates for quintiles III and IV are systematically lower than they should be to give a smooth trend. This arises from the

TABLE 85

Weighted mean quintile death rates, per 1000, at ages, for outdoor occupations

QUINTILE	AGE GROUPS							
	15–19	20–24	25–34	35–44	45–54	55–64	65–74 (see p. 303)	75 and over (see p. 303)
I	1.27±0.06	2.45±0.07	3.57±0.06	6.02±0.08	11.29±0.13	23.49±0.23	52.58±0.46	163.00±1.35
II	1.63±0.04	3.16±0.06	4.05±0.05	7.38±0.07	13.97±0.12	29.30±0.21	67.62±0.47	174.93±1.43
III	1.29±0.06	2.97±0.08	4.51±0.07	6.34±0.09	11.64±0.13	23.88±0.21	56.39±0.40	162.20±1.01
IV	1.46±0.04	2.60±0.05	3.61±0.05	6.17±0.07	10.77±0.10	22.70±0.17	52.91±0.33	177.25±0.97
V	1.83±0.04	2.64±0.05	3.44±0.04	6.21±0.07	12.71±0.12	29.31±0.24	77.37±0.60	211.07±2.08

TABLE 86

Absolute figures for populations and deaths, outdoor occupations

QUINTILE	AGE GROUPS															
	15–19		20–24		25–34		35–44		45–54		55–64		65–74		75 and over	
	*A**	*B**	*A*	*B*	*A*	*B*	*A*	*B*	*A*	*B*	*A*	*B*	*A*	*B*	*A*	*B*
I	190,839	242	221,973	543	508,029	1,815	428,373	2,580	301,530	3,404	193,260	4,539	108,576	5,709	34,227	5,579
II	517,596	842	370,245	1,169	817,764	3,310	633,945	4,679	455,163	6,357	285,429	8,363	131,832	8,914	32,241	5,640
III	159,309	205	186,171	552	424,272	1,913	371,343	2,355	317,997	3,700	235,812	5,585	152,049	8,574	60,666	9,840
IV	442,968	609	406,686	1,057	699,510	2,522	575.007	3,547	481,275	5,183	340,461	7,727	212,643	11,252	69,984	12,405
V	464,922	849	450,057	1,187	815,244	2,805	650,997	4,040	418,581	5,319	229,857	6,738	89,274	6,907	17,463	3,686

* See footnote to table 82 above

inclusion in these two quintiles of the two healthiest occupations involving large numbers of persons, namely 32, farmers, graziers, farmer's sons (in quintile III), and 33, agricultural laborers, farm servants (in quintile IV). The great healthfulness of the farm environment apparently a good deal more than offsets the effects of hard work. And probably occupation group 32 is in too high a quintile anyway. The British farmer *himself* probably does not work with anything approaching the severity that the American farmer *himself* does, considering both cases statistically. And in placing this occupation our group of 9 probably were influenced by their knowledge of the American farmer's habits.

Taking the data as a whole all groups except one (age 25 to 34) show some upward general trend in passing from quintile I to V. This trend is slight

TABLE 87

Differences in mortality rates between Quintile I and Quintile V. Outdoor occupations

AGE GROUP	DIFFERENCE V − I	RATIO OF DIFFERENCE TO ITS PROBABLE ERROR	PERCENTAGE INCREASE (OR DECREASE) IN MORTALITY RATE IN PASSING FROM QUINTILE I TO V
			per cent
15–19	+0.56±0.07	8.0	+44.1
20–24	+0.19±0.09	2.1	+7.8
25–34	−0.13±0.07	1.9	−3.6
35–44	+0.19±0.11	1.7	+3.2
45–54	+1.42±0.18	7.9	+12.6
55–64	+5.82±0.33	17.6	+24.8
65–74*	+24.79±0.76	32.6	+47.1
75 and over	+48.07±2.48	19.4	+29.5

* See page 303 regarding this and the next age group.

in the early age groups (except 15 to 19), as was the case with the indoor occupations, but becomes marked after age 45.

Just as in the case of the indoor occupations, no one can possibly deny that the occupations in quintile V require in their pursuit a great deal higher expenditure of physical energy, on the average, than do those in quintile I. Let us examine the differences between these extremes, in respect of mortality. The differences, with their probable errors, are set forth in table 87.

The figures in the difference column of this table are shown graphically in figure 53. It is apparent at once that the outdoor occupations tell the same story as the indoor. In the earlier age groups it makes little difference in the specific mortality rates whether people are engaged in physically

heavy or light occupations. But after age 45 is past the toll is increasingly heavier in the occupations which involve strenuous physical labor.

The results throw interesting light on the general biology of death. The males in this study all enter (in the actuarial sense) at ages at or above 15 years. They are a biologically selected lot in other words, being the *survivors* at age 15 and over of the cohorts from which the infant and childhood mortality have taken heavy toll. Having arrived at these ages and

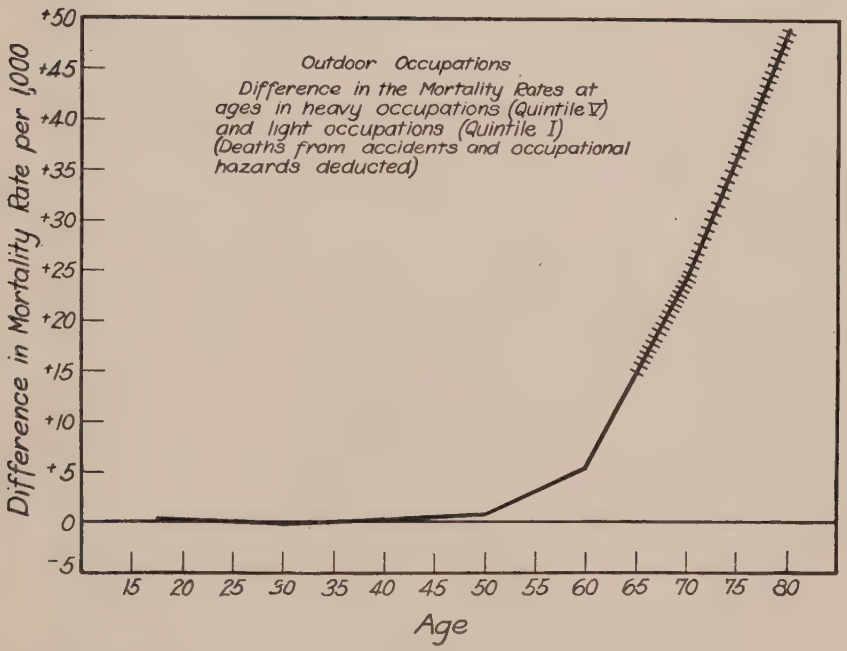

FIG. 53. DIFFERENCES IN THE MORTALITY RATES AT AGES BETWEEN HEAVY OCCUPATIONS (QUINTILE V) AND LIGHT OCCUPATIONS (QUINTILE I). OUTDOOR OCCUPATIONS

The line is crossed from age 65 on to indicate the uncertainty as to its true position at the advanced ages.

passed through this selective process, our present results show that the large environmental factor which is expressed in occupation, makes little difference in their mortality rates for the next 25 to 30 years. Their innate, inherited *ability to live* surpasses, in power to influence the situation, the effect of such great environmental differences as are expressed by our occupation quintile groups I and V. But after age 45 is past the environmental factors make their influence felt in increasing degree.

There is another phase of the environment problem which is of interest. In figures 54 and 55 the age specific death rates for our "chiefly indoor" and "chiefly outdoor" groups of occupations are compared; for light occupations (quintile I) in figure 54, and for heavy occupations (quintile V) in

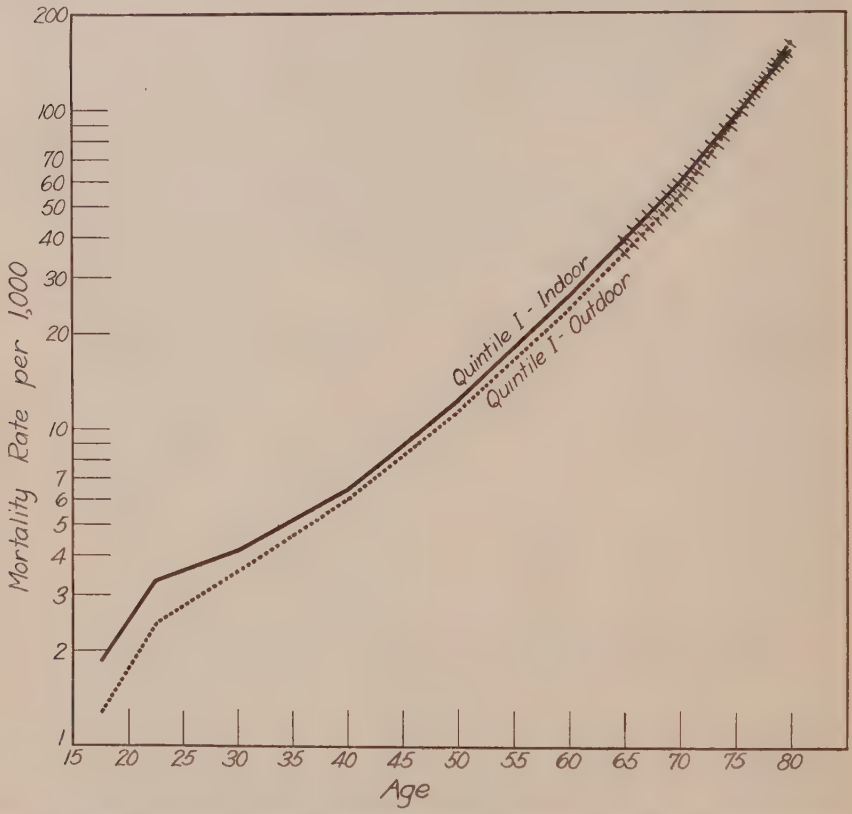

FIG. 54. AGE SPECIFIC DEATH RATES OF MALES ENGAGED IN LIGHT OCCUPATIONS (QUINTILE I) WHICH ARE (*a*) CHIEFLY INDOOR AND (*b*) CHIEFLY OUTDOOR IN THEIR ENVIRONMENT

In this and the next diagram the lines are crossed from age 65 on to indicate the uncertainty as to the true values of the death rates at those ages.

figure 55. These diagrams are the plots of the first and last *rows* of figures in tables 81 and 85.

These data show, in a striking way, the superior healthfulness of outdoor as compared with indoor work. In the case of the light occupations (fig. 54) the indoor death-rate line lies above the outdoor till about age 75 and

there the values are uncertain anyhow. In the heavy occupations the lines cross about 10 years earlier. Enormous improvements have been made during the last 50 years in the sanitary conditions under which indoor labor is performed. Especially has this been true in England. But there

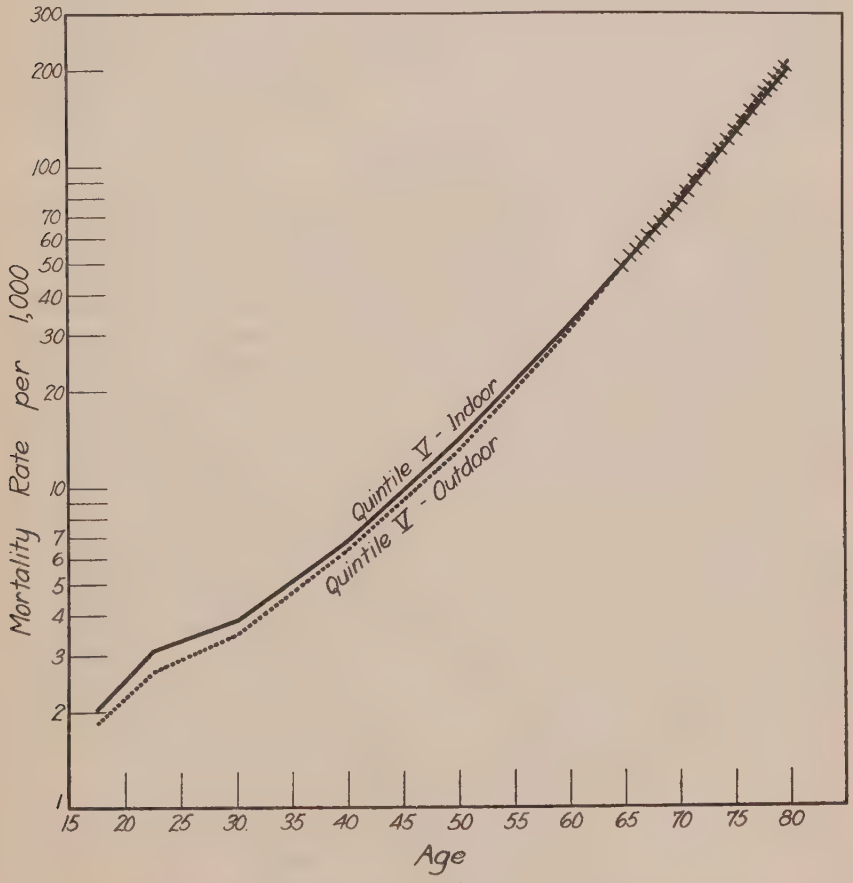

FIG. 55. AGE SPECIFIC DEATH RATES OF MALES ENGAGED IN HEAVY OCCUPATIONS (QUINTILE V) WHICH ARE (*a*) CHIEFLY INDOOR AND (*b*) CHIEFLY OUTDOOR IN THEIR ENVIRONMENT

is still more to be done. A part of this will consist in putting into practice, more completely than has yet been done, well known and fully understood methods of making factories and shops more sanitary and healthful places in which to work. But perhaps if all this were done the two lines of figures

54 and 55 would even then not coincide, which might reasonably be taken to mean that we still had something to learn about the relation of housing (in the broadest sense) to health and well being.

It will be noted that in both figures 54 and 55 the outdoor and indoor lines are farther apart between ages 15 and 30 than at any other part of their course. This is to be attributed, as examination of the figures shows, mainly to the relatively heavy mortality from tuberculosis in these ages among persons employed chiefly indoors.

SOCIAL CLASSES

A point of criticism which will occur to anyone who has followed the discussion up to this point is this: Can the differences in mortality as we pass from occupations involving little expenditure of physical energy (quintile I) to those involving great expenditure (quintile V), be attributed solely, or even chiefly to this factor? Are they not rather due to the general environmental differences implicit in social class and economic distinctions? Some, at least, of the occupations in quintile I, for example, are those practiced by the professional classes, with relatively high incomes and standard of living, with which reduced mortality is, on superficial examination at any rate, supposed to be invariably associated.

It is evident that this point wants careful examination and consideration. Fortunately the original report gives data on the grouping of the occupations into social classes by the Census. But in some cases subdivisions of a large occupational group are put into different social classes, and the social classification used is not a logically self-consistent scheme. Still it does furnish some basis for discussion.

The social classes adopted by the Census authorities are:

Social class I. Upper and middle classes
II. Intermediate, excluding unoccupied
III. Skilled workmen
IV Intermediate
V. Unskilled workmen
VI. Textile workers
VII. Miners
VIII. Agricultural laborers

It will be noted at once that the classification changes its basis abruptly after Class V is passed. Up to that point it is essentially a *social* classification; beyond that point it is not essentially social but *occupational*. We are consequently restricted in our present consideration of the matter to classes

I to V. But a still further restriction of the available data results from the fact that most of the occupations which the census authorities put in social class V, are occupations which are chiefly outdoor in their environment. Consequently, in order to get a statistically respectable body of reasonably homogeneous material for discussion we are thrown back mainly upon social classes I to IV inclusive, for chiefly indoor occupations.

TABLE 88

Reference numbers of the indoor occupations put in the first six social classes by the Census

SOCIAL CLASS					
I	II	III	IV	V	VI
1	9	11	51	49	97
3	10	45	52	84	98
4	44	46	53	85	99
5	63	47	54	90	100
6	64	48	55		101
7	75	50	56		102
8	93	60	57		103
13	94	61	58		104
14	105	62	76		
15	112	65	77		
16	113	66	80		
18	114	71	81		
67	116	73	82		
83	117	74	86		
	118	88	87		
	119	89	91		
	121	92	106		
	124	95	107		
	130	96	115		
		108	122		
		109	123		
		110	127		
		120	128		
		125	132		
		126			
		129			

The social classification of our indoor occupations, as extracted from the original Report, is shown in table 88.

The relation between social class position and quintile position on our scheme is shown in table 89 for indoor occupations.

The result is what might be expected. The very basis of social class distinctions is, in good measure, the amount of physical labor which a

person performs. Consequently the occupations in social class I are preponderantly of quintile I in energy expenditure, while those of social class V fall in quintiles III, IV and V.

The situation depicted in table 89 shows at once the difficulty of the problem confronting us. What we wish to do is to disentangle the effects upon mortality of social class from those of physical energy expenditure. But these two variables, because of an underlying fact of social psychology are very firmly tied together. And ordinary analytical procedures, such as partial correlation, cannot be applied in this instance because we do not know the quantitative values of the scale units for either social classes or energy quintiles. Whatever procedure is adopted must always keep this point in mind. It, in fact, rules out the simplest and most direct of all methods of approaching a set of data, namely, the graphic. After a good

TABLE 89

Frequency of indoor occupations by social class and quintile position

SOCIAL CLASS	OCCUPATION QUINTILES					TOTALS
	I	II	III	IV	V	
I	11	2	1			14
II	7	9	3			19
III	1	4	6	11	4	26
IV		2	3	6	13	24
V			2	1	1	4
VI		2	4	1	1	8
Totals.....	19	19	19	19	19	95

deal of study over the problem, however, we have worked out a method of approach which, as it seems to us, makes quite clear and definite the real truth of the matter.

As a first step we calculated, for the 95 indoor occupations, the death rates specific for age, energy quintile, *and* social class. This was done for all age groups, but as we have already seen that the occupational energy expenditure factor does not make any sensible effect upon mortality till after age 40 is passed, and since an examination of the social class mortality rates show that the same thing holds there, we shall present here and discuss only the data from the last four age groups, 45 to 54, 55 to 64, 65 to 74, and 75 and over. For reasons already stated no stress can be laid on the last two of these age groups. The data are only included for the sake of completeness. No conclusion can at present be drawn from them. The specific death rates for these ages are set forth, with their probable

errors, in tables 90 to 93 inclusive. In these tables only the first five social classes are included, for the reason, already discussed, that the basis of classification is changed when class VI is reached.

From the most superficial examination of tables 90 and 91 one notes the following facts: As we pass from lower to higher occupation quintiles (left

TABLE 90

Weighted mean death rates, by social class and occupation quintiles, for the age group 45–54. Indoor occupations

SOCIAL CLASSES	OCCUPATION QUINTILE				
	I	II	III	IV	V
I	11.90 ±0.13	12.64 ±0.31	11.76±0.42		
II	12.47 ±0.24	15.51 ±0.17	14.58±0.22		
III	17.68*±0.80	14.40 ±0.28	15.26±0.23	14.73±0.19	12.58±0.14
IV		16.15†±0.88	10.26±0.41	13.07±0.26	14.35±0.19
V			11.75±0.67	11.65±1.00	15.08±0.24

* This includes but one occupation, no. 110 (hair-dressers, wig-makers) which exhibits an abnormally high mortality from a number of causes. Some of these seem probably the result of special occupational hazards, but we have no specific evidence to warrant deducting them.

† This excessively high rate is mainly due to tuberculosis which is unquestionably an occupational hazard not anything like completely allowed for in this case, since all the individuals in this group are in occupations 106 and 107 (manufacture of hats, straw, cloth, or felt).

TABLE 91

Weighted mean death rates, by social class and occupation quintiles, for the age group 55–64. Indoor occupations

SOCIAL CLASSES	OCCUPATION QUINTILE				
	I	II	III	IV	V
I	25.66 ±0.25	31.31±0.64	29.99±0.75		
II	26.51 ±0.43	30.00±0.29	29.73±0.42		
III	34.22*±1.67	29.86±0.51	30.24±0.10	29.61±0.36	28.25±0.26
IV		26.12±1.43	26.99±0.92	28.63±0.49	32.26±0.37
V			24.45±1.23	28.50±2.23	32.76±0.43

* See footnote to the preceding table.

to right along the rows of the tables) the death rates specific for *both* social class and energy expenditure, tend to *increase* rather markedly. This is what would be expected from results already attained. It means that in any social class greater physical strenuousness of occupation has associated with it greater mortality. When, on the other hand, we pass

from the highest social class (I) to the lower (down the columns of the tables) there is no such definite or marked tendency for the doubly specific mortality rates to increase. On the contrary, in many cases they plainly decrease.

These facts are of real importance. They mean that social class, and the general environmental factors which it connotes apart from energy expenditure in occupation, have distinctly *less* influence upon the rate of

TABLE 92

Weighted mean death rates by social class and occupation quintiles, for the age group 65–74. Indoor occupations

(*The data of this table must be viewed with great caution. See page 303*)

SOCIAL CLASSES	OCCUPATION QUINTILE				
	I	II	III	IV	V
I	58.59±0.53	67.71±1.42	75.77±1.54		
II	63.02±0.92	67.87±0.61	63.37±0.87		
III	65.48±3.57	66.74±1.02	69.78±0.63	65.07±0.78	68.98±0.58
IV		65.53±3.25	60.73±2.16	68.89±1.09	74.74±0.83
V			53.06±2.54	63.44±5.39	77.81±0.98

TABLE 93

Weighted mean death-rates, by social class and occupation quintiles, for the age group 75 and over. Indoor occupations

(*The data of this table must be viewed with great caution. See page 303*)

SOCIAL CLASS	OCCUPATION QUINTILE				
	I	II	III	IV	V
I	146.87±1.42	184.76± 4.35	193.28±3.87		
II	174.70±2.75	182.76± 1.73	168.21±2.47		
III	162.96±9.59	169.60± 2.55	182.67±1.93	183.04± 2.42	181.70±1.82
IV		187.88±10.26	179.62±7.48	201.80± 3.37	205.24±2.70
V			201.58±9.08	222.22±23.37	225.06±3.39

mortality in occupied males after age 45 than does the physical strenuousness of occupation. Let us see whether a numerical expression of these facts can be worked out, which shall be independent of, in the sense of not being vitiated by, the fact that it is not possible exactly to quantify the abscissal scales of either energy expenditure or social class. It is believed that the following procedure roughly meets this requirement, but with sufficient precision to establish the point, which is all that we are concerned to do.

The process, in brief, is to take the mean differences between the specific death rates in the table for all possible pairs, first by rows, and then by columns, having regard for the signs of differences throughout.

An example will make the case clearer. Consider social class III, age 65 to 74 (table 92) purely from the standpoint of method. The death rates in this row are, for the several quintiles, as follows: 65.48, 66.74, 69.78, 65.07, 68.98. These figures show the progression of mortality rates for persons in social class III, with advancing quintile position in energy expenditure. We have the following system of possible differences within this row:

IN PASSING		DEATH RATE DIFFERENCE	
From quintile	To quintile	+	−
I	II	1.26	
I	III	4.30	
I	IV		0.41
I	V	3.50	
II	III	3.04	
II	IV		1.67
II	V	2.24	
III	IV		4.71
III	V		0.80
IV	V	3.91	
Totals.....		+18.25	−7.59

Net total = 18.25 − 7.59 = +10.66

Now suppose differences to be taken in this same way for all the *rows* of each table and the results summed, for the whole table. The results will be those in the first column of table 94, headed "total net difference" (by quintile position). The other half of table 94 is made in the same way except that the differences are taken by columns instead of rows.

The philosophy of this procedure is this: if the general trend of the death rates in a row or column is *upward* (from left to right or top to bottom) the total net and average differences of table 94 are bound to be *positive*, and larger as the upward trend becomes more marked, or steeper. Conversely, if the trend of the death rates is *downward*, the differences are bound to be *negative*, and larger the steeper the trend. If there is no trend of the rates in the column or row, as a whole, the net differences will be zero. These relations will be true whatever the abscissal position of the death rates, provided only that social class or quintile positions are in progressive order, which is the case.

It is seen from this table that the differences by quintile position, indicating the effect of physical energy expenditure upon mortality, are all *positive* with a single exception. In that case (45 to 54, all data) two highly abnormal rates in low quintiles are included, though it is certain that they do not conform to the standards set for our data in the beginning. On the other hand, all the differences by social class are *negative* with two exceptions. Of these the first (45 to 54, all data) again includes the abnormal rates already mentioned. In the other (75 and over) it is to be recalled that the rates here cannot be regarded as reliable.

Altogether we believe that the data demonstrate, for indoor occupations, that *the adverse effect of increasing energy expenditure upon mortality at ages*

TABLE 94

Differences and their trends, in specific death rates by (a) quintile position, and (b) social class. Indoor occupations

AGE GROUP	BY QUINTILE POSITION			BY SOCIAL CLASS		
	Total net difference	Number of pairs	Average difference	Total net difference	Number of pairs	Average difference
45–54 (all data)	−11.73	25	−0.47	+11.14	25	+0.45
45–54 (with asterisked rates of table 90 omitted)	+12.79	18	+0.71	−5.75	20	−0.29
55–64 (all data)	+27.40	25	+1.10	−20.88	25	−0.84
55–64 (with asterisked rate of table 91 omitted)	+46.30	21	+2.20	−37.15	23	−1.62
65–74 (all data)*	+131.01	25	+5.24	−75.61	25	−3.02
75 and over (all data)	+302.90	25	+12.12	+249.49	25	+9.98

* Note the caution to be observed regarding this and the following age group, page 303.

after 45 is not the result of environmental influences associated with social class distinctions to any significant degree, but is directly and unequivocally the result of the physically hard work itself.

Let us now turn to the outdoor occupations. The social classification of these occupations, as extracted from the original report, is given in table 95.

The relation between social class and occupation quintile for these occupations is shown in table 96.

It is apparent, just as in the indoor occupations, that there is a marked association between quintile position and social class in the outdoor occupations. It however is apparently somewhat less regular and definite than in the former case.

TABLE 95

Reference numbers of the outdoor occupations put in the several social classes by the Census

SOCIAL CLASS						
I	II	III	IV	V	VII	VIII
17	27	12	2	21	36	33
	32	19	34	22	37	
	111	20	35	25	38	
		23	42	28	39	
		24	43	29		
		26	59	30		
		68		31		
		69		40		
		70		41		
		72		78		
				79		
				131		

TABLE 96

Frequency of outdoor occupations by social class and quintile position

SOCIAL CLASS	OCCUPATION QUINTILE					TOTAL
	I	II	III	IV	V	
I	1					1
II		1	2			3
III	4	3	2	1		10
IV	2		1	2	1	6
V		3	3	4	2	12
VI						
VII					4	4
VIII				1		1
Totals...	7	7	8	8	7	37

TABLE 97

Weighted mean death rates, by social class and occupation quintile for the age group 45–54. Outdoor occupations

SOCIAL CLASS	OCCUPATION QUINTILE				
	I	II	III	IV	V
I	12.56±0.45				
II		13.47±0.63	11.52±0.16		
III	14.90±0.24	12.84±0.14	12.78±0.30	8.33±0.36	
IV	8.31±0.16		13.36±0.67	14.10±0.24	13.85±0.96
V		16.08±0.22	9.09±0.38	17.02±0.30	19.73±0.33

Death rates specific for *both* social class and energy expenditure, as indicated by quintile position, are given for the outdoor occupations in tables 97 to 100 inclusive, for the four age groups from 45 on. Here as before we can only make use of the first five social classes, because VII and VIII are upon a wholly different basis than I to V.

Unfortunately the number of outdoor occupations is so small, and there is such extreme variation in the mortality associated with them, partly owing to the complication already mentioned that outdoor life is *per se* healthy, that the results are rather irregular, both in respect of energy expenditure and of social class. The outstanding abnormality in the results arises from social class III. Consequently the difference table has been made up to show the results both with and without the inclusion of this social class. This table, made on exactly the same plan as table 94 for indoor occupations is given in table 101 for outdoor occupations.

TABLE 98

Weighted mean death rates, by social class and occupation quintile, for the age group 55–64. Outdoor occupations

SOCIAL CLASS	OCCUPATION QUINTILE				
	I	II	III	IV	V
I	26.91±0.90				
II		27.38±1.20	23.47±0.25		
III	31.37±0.46	27.33±0.25	24.83±0.50	24.12±0.79	
IV	18.45±0.27		24.74±1.10	29.83±0.42	36.61±1.81
V		33.83±0.41	22.21±0.77	33.29±0.50	33.69±0.58

From this table it is clear, in the first place, that even when the death rates are specific for social class the average trend of the differences in mortality as we pass from lower quintiles (less energy expenditure) to higher quintiles (more energy expenditure) is in general upward. The differences by quintiles are positive except in the last group (75 and over) where the rates in general cannot be relied upon. In the other age groups, if the aberrant social class III be omitted, the differences by social class, in comparison with those by occupation quintiles, indicate that, just as in the case of indoor occupations, the conclusion that increased energy expenditure in occupation increases the rate of mortality after age 45, is not vitiated by the fact that social class distinctions are correlated with energy expenditure in occupation.

The just conclusion from the whole case appears to us to be that the data from outdoor occupations are too meager to permit any precise measure-

TABLE 99

Weighted mean death rates, by social class and occupation quintile, for the age group 65–74. Outdoor occupations

(The data of this table must be viewed with great caution. See page 303)

SOCIAL CLASS	OCCUPATION QUINTILE				
	I	II	III	IV	V
I	48.09±1.85				
II		74.26±2.70	55.74±0.45		
III	70.91±0.99	63.57±0.54	56.89±1.09	61.67±2.18	
IV	45.25±0.52		58.07±2.17	66.05±0.86	80.03±3.72
V		77.24±0.95	63.37±1.80	74.53±1.03	65.64±1.29

TABLE 100

Weighted mean death rates, by social class and occupation quintile, for the age group 75 and over. Outdoor occupations

(The data of this table must be viewed with great caution. See page 303)

SOCIAL CLASS	OCCUPATION QUINTILE				
	I	II	III	IV	V
I	132.48±6.68				
II		227.12±8.08	159.83±1.09		
III	195.09±2.79	159.32±1.60	158.40±3.48	191.04±8.36	
IV	152.18±1.57		173.80±5.87	181.15±2.68	166.67±10.70
V		218.29±3.30	214.10±5.77	229.64±3.31	176.04± 4.92

TABLE 101

Differences, and their trends, in specific death rates by (a) quintile position, and (b) social class. Outdoor occupations

AGE GROUP	BY QUINTILE POSITION			BY SOCIAL CLASS		
	Total net difference	Number of pairs	Average difference	Total net difference	Number of pairs	Average difference
45–54 (all data)	+14.52	19	+0.76	+13.27	16	+0.83
45–54 (Social Class III omitted)	+34.29	13	+2.64	+2.30	7	+0.33
55–64 (all data)	+42.07	19	+2.21	+7.53	16	+0.47
55–64 (Social Class III omitted)	+66.32	13	+5.10	−3.99	7	−0.57
.....	.....	.....	.....	.....	.....	.....
65–74 (all data)*	+35.76	19	+1.88	+35.68	16	+2.23
65–74 (Social Class III omitted)	+70.16	13	+5.40	+9.49	7	+1.36
75 and over (all data)	−140.73	19	−7.41	+286.52	16	+17.91
75 and over (Social Class III omitted)	−127.68	13	−9.82	+177.27	7	+25.32

* Note the caution to be observed regarding this and the next higher age group, page 303.

ment of the relative influence of heavy physical labor, on the one hand, and unfavorable general environmental circumstances associated with membership in the lower social classes, on the other hand, in increasing mortality after age 45. But so far as the data warrant inference, they seem to be consistent, allowing for their meagerness and roughness, with those for indoor occupations.

SUMMARY AND DISCUSSION

The essential result of this long and tedious analysis may be stated in a few words:

Taking as a basis of analysis what may be fairly regarded as the most comprehensive and accurate statistics of occupational mortality now in existence, it appears that in occupied English males, there is a direct and positive relation between the magnitude of the age specific death rates from age 40 to 45 on, and the average expenditure of physical energy in occupation, after accidental deaths, and deaths directly resulting from the hazards of each of the several occupations have been deducted in so far as the official statistics by causes of death permit such deductions. This relation is of the sort that associates high mortality with hard physical labor. This relationship prevails whether the labor is performed chiefly indoors or chiefly outdoors. It is not primarily to be attributed to the general environmental factors connoted by social class distinctions, which are themselves correlated with average energy expenditure in occupation. (This last conclusion appears from the present data reasonably certain for indoor occupations, and probable for outdoor occupations.) Before age 40 is attained, it makes no difference in the rate of mortality whether the occupation involves light or heavy physical labor. After roughly age 40 to 45 it appears that a man shortens his life, by definite amounts, in proportion as he performs physically heavy labor.

So far as concerns *extreme* energy expenditure there is nothing novel about this result. It has long been recognized to be a fact. The following passage from Ross[7] (pp. 84, 85) regarding the Chinese is a sufficient example.

> Still, it is obvious that in certain occupations men are literally killing themselves by their exertions. The treadmill-coolies who propel the stern-wheelers on the West River admittedly shorten their lives. Nearly all the lumber used in China is handsawed, and the sawyers are exhausted early. The planers of boards, the marble polishers, the brass filers, the cotton fluffers, the treaders who work the big rice-polishing pestles are building their coffins. Physicians agree that carrying coolies rarely live beyond forty-five or fifty

[7] Ross, E. A., *The Changing Chinese*, New York (Century Co.), 1912.

years. The term of a chair-bearer is eight years, of a rickshaw runner four years; for the rest of his life he is an invalid. Moreover, carriers and chair-bearers are afflicted with varicose veins and aneurisms because the constant tension of the muscles interferes with the return circulation of the blood. A lady physician in Fokien who had examined some scores of carrying coolies told me she found but two were free from the heart trouble caused by burden-bearing.

What the present study may perhaps justly claim to have done is to have rendered it extremely probable that the same unfortunate relationship between hard work and duration of life shown in these extreme cases also holds in graded degrees after middle life for nearly every walk of life. The hypothesis stated by Pearl in *The Biology of Death*, quoted at the beginning of this study, seems to be definitely supported by the facts of occupational mortality.

Confirmation of the results of this study is, in a general though not very precise way, afforded by the recent study by Kopf and Van Buren[8] of the mortality experience of the leading industrial insurance companies. The mortality of the insured wage earners is higher than that of the general population at practically all ages.

That physical toil is *per se* a factor in tuberculosis mortality has long been recognized. Recently Arnould[9] has discussed this matter in a penetrating way. He points out that the fall in the tuberculosis death rate in recent years has been relatively greater for men than for women, and that in consequence the mortality among women from this disease has become equal to or even greater than that of men, in some groups, national or economic. The relative increase in the death rate among women coincides with the relative increase in the number of women in industry. He believes that it is work *per se* that is responsible for the increased liability to tuberculosis, and not primarily factory conditions, and cites as evidence of this contention the effect of agricultural work. Where women have largely taken over the cultivation of the soil, while the men go into industry, tuberculosis among the women has increased to a point which brings their mortality rate nearly to that of males. In the country in France, where women are, as a rule, more laboriously employed than the women of the towns, Arnould states that the death rate is higher.

Farr[10] (pp. 392–411), as would be expected, discussed occupational mortality with his usual wisdom and sagacity, and his treatment of the

[8] *The Mortality Experience of Industrial Policy-holders 1916–1920*, New York, 1923, 150 pp.

[9] Arnould, E., La mortalité tuberculeuse du sexe féminine, *Rev. d. l. Tuberc.*, T. 3, p. 141, 1922.

[10] Farr, W., *Vital Statistics*. (Edit. Humphreys), London, 1885.

subject may be read today with great profit by any vital statistician. But it has little specific bearing upon the particular problem with which the present paper has to do.

It is apparent that the results of this study have a social bearing. We do not propose to enter upon any extended discussion of this aspect of the matter here, as it falls outside our present purpose, and furthermore appears to demand an amount of wisdom far beyond our limited store. But we cannot refrain from pointing out that the already sufficiently difficult task of public health to improve the mortality in ages after middle life, is shown by this study to be made much more difficult by an economic structure of society which condemns a great many men of advanced ages to perform physically hard labor or starve.

CHAPTER XII

The Longevity of the Parents of the Tuberculous and the Cancerous[1]

There is a widespread idea that cancer is especially likely to attack constitutionally strong and healthy individuals. This idea has found expression many times in medical literature (*cf.* Bauer's[2] excellent review). It appears not to be grounded upon the results of any particular statistical investigations, but rather to express what is thought, at any rate by many, to be the general experience of practitioners.

Similarly, tuberculosis is generally thought to attack the constitutionally weak. Tuberculous stocks are held to be generally weak, in respect of physique, resistance to disease, etc. It has been asserted that some of the evidence regarding the inheritance of the tuberculous diathesis strongly indicates the general constitutional inferiority of such stocks.

It is the purpose of this chapter to present some new evidence bearing upon these questions. Perhaps the best single measure of the constitutional soundness—strength or weakness—of the individual is his duration of life. This datum is a single numerical measure of the combined effect on the organism of all factors, hereditary and environmental in the broadest sense of these words, which influence its life history. The sounder one's constitution the more and graver the environmental accidents which he will be able to pass through successfully. There can be no question that, considering the matter statistically, persons with what the medical man would call poor and feeble constitutions die on the average at an earlier age than do those who have what general medical experience would class as sound or "strong" constitutions.

It would be idle to attempt to test directly the question of the general constitutional soundness of either the tuberculous or the cancerous, by a study of the ages at death of the affected individuals themselves, for the reason that each of these diseases has a characteristic time of attack, in the whole life history of the individual. Tuberculosis is primarily a disease of

[1] The paper on which this chapter is based first appeared under the title, "The age at death of the parents of the tuberculous and the cancerous," in *Amer. Jour. Hyg.*, vol. 3, pp. 71–89, 1923. It has only been modified here to a slight extent.

[2] Bauer, J. *Die konstitutionelle Disposition zu inneren Krankheiten*, Zweite Aufl. Berlin (Springer), 1921, xi + 650 pp., 8vo.

early life. Cancer is primarily a disease of more advanced ages. Cancerous persons die at a fairly high average age. But this merely means that, broadly speaking, the only persons who ever have cancer are those in, or past, middle life.

But there is another way of getting at the matter. The constitution of the individual, in the sense that I am here using the word, is undoubtedly inherited, and so is its index, duration of life (*cf.* Bauer, *loc. cit.*, Pearl [3]). We may then get some indication of the constitutional superiority or inferiority of the stock from which the cancerous or tuberculous individual comes, by examining the ages at death of the individual's immediate ancestors. In the present chapter the following question is put: Does the mean age at death of the parents of the tuberculous (or the cancerous) differ sensibly from the mean age at death of the parents of the non-tuberculous (or non-cancerous), the parents themselves being non-tuberculous (or non-cancerous)? If any weight is attached to the prevailing opinions of clinicians, it would be expected that the parents of the tuberculous would have a *lower* mean age at death than the parents of the non-tuberculous, and that the parents of the cancerous would have a *higher* mean age at death than the controls. Bauer (*loc. cit.*, p. 82) makes, without presentation of any evidence, this statement regarding the parents of the cancerous. "Systematische einschlägige Untersuchungen, die ich gemeinsam mit Frl. Kerti begann, bestätigten insbesondere immer wieder die auffallende Langlebigkeit der Eltern von Krebskranken."

MATERIAL AND METHODS

The material for this study was extracted from the Family History Records of the Department of Biometry and Vital Statistics. These Family History Records consist of elaborate detailed original pedigrees, collected by a staff of trained eugenic field-workers. In order that a clear understanding may be had of the significance of this material, some account of its history is desirable. Some five years ago at the request of the National Tuberculosis Association, and with its financial support at the outstart, and that of the Russell Sage Foundation and the Commonwealth Fund later, the writer embarked upon a comprehensive investigation of the factors involved in the etiology of tuberculosis, with special reference to the genetic elements in the case. The plan of the work, in outline, was as follows: It was

[3] Pearl, R., A note on the inheritance of duration of life in man, *Amer. Jour. Hyg.*, vol. 2, pp. 229–233, 1922.

Pearl, R., *The Biology of Death*, Philadelphia (J. B. Lippincott Co.), 1922.

proposed to collect a large number of much more detailed and elaborate family histories of tuberculous persons than any that had hitherto been compiled, doing this by means of field- or social-workers, carefully trained for this work. These field-workers visit the families of individuals whom it is desired to investigate, and get by personal interview the information to be set down in the histories. Proceeding in the matter in an entirely objective way and without preconceptions, it was felt to be essential, if any critical results were to be obtained, to get the most elaborate and critically exact records possible about the environmental situation, the habits of life, the health history, the racial stock, anthropological characteristics, exposure to tuberculosis, etc., of all the members of the family group.

The histories have, from the first, been divided into two groups. In one set the history starts with an individual, the propositus, who is known to have clinically manifest tuberculosis. In the other set each history starts with a propositus who is definitely known *not* to have tuberculosis in any form. The initial individuals for these two sets of histories have been obtained in the following ways. The tuberculous individuals were taken at random (except for race stock) from among those persons who were registered with the Baltimore City Health Department as having active tuberculosis, under the law which makes this a reportable disease; or from those persons registered with the Phipps Tuberculosis Clinic of the Johns Hopkins Hospital, a free dispensary clinic. The non-tuberculous individuals were taken at random (except for race stock), either from among those persons who had, for some trivial offence (such as, for example, playing baseball in a vacant lot, etc.), been before the Juvenile Court and were known not to be tuberculous, or from the patients registering at the General Dispensary of the Johns Hopkins Hospital, and known not to be tuberculous.

Starting from the propositus the procedure is the same in both sets of histories. Through the work of the field-workers the family history is traced, both in respect of the ancestry and their collaterals, and in respect of descendants and their collaterals. The histories record identically the same kind of facts in the two groups in every respect. The only difference is that in the one case they start with an individual known to be tuberculous, and in the other case they start with an individual known not to be tuberculous. The same questions are asked and the same facts recorded for all the individuals in the family tree in both sets. The collection of this material has now been completed. It may be well to state, as indicating in some degree the magnitude of the investigation, that detailed records of the life-history of approximately one hundred and thirty thousand individuals, comprised in about four hundred and twenty-five family trees, have been collected.

Every critical safeguard of the accuracy of the ultimate individual records that the writer has been able to think of has been thrown about this work. No fact is entered in a history finally until it has been corroborated by the independent testimony of at least two persons acquainted with the individual in question. The material has all been taken from one socially and economically homogeneous group of the population of the city of Baltimore, namely, what might inclusively be called working-men's families. By the employment of field-workers speaking a variety of foreign languages it has been possible to get representation in the histories of the different foreign race stocks in about the proportion that they are represented in the total population of the city of Baltimore, and in this way there has been no racial discrimination in the study. The only exception to this is that the negro has not been included, owing to the difficulty—indeed, impossibility—of getting accurate genealogical information about negro families. These people do not know accurately about their own ancestors.

The elaborate records which are taken regarding the environmental surroundings of the persons in the histories, demonstrate that they may be regarded as forming a homogeneous group in this respect. From these Records were extracted for the present study the following items:

1. The age of each *tuberculous* person.
2. Whether the person was dead or living.
3. The age of (*a*) the father, (*b*) the mother of the person.
4. Whether (*a*) the father and (*b*) the mother were living or dead.

The same four items were extracted for each person known to have, or to have had, *cancer* in any form.

The records so extracted were transferred to Hollerith cards, and the subsequent tabulations were made on Tabulating Machine Company equipment.

The next step, which proved of much interest, was to get a proper control group. At first thought the thing to do might be supposed to be to take from the Records a random sample, equal in size to the tuberculous sample, of non-tuberculous individuals (and non-cancerous individuals) and extract from them the same four items as had been taken for the tuberculous and cancerous. In point of fact this was done, and forms the so-called Random Control which appears in the tables farther on.

It was quickly apparent, however, that this furnished no proper control group, for a reason which was obvious upon a little consideration. Tuberculosis is a disease of early life. Persons dying of the disease die at a relatively low average age (*cf.* Pearl and Bacon[4]). A random sample of non-

[4] Pearl, R., and Bacon, A. L., Biometrical studies in pathology. I. The quantitative relation of certain viscera in tuberculosis, *Johns Hopkins Hospital Repts.*, vol. 21, fasc. 3, pp. 157–230, 1922.

tuberculous persons will die at a considerably higher average age. Now since it is well known (Beeton and Pearson,[5] Bell,[6] Pearl, *loc. cit.*) that there is a definite and significant correlation between parent and offspring in respect of age at death, it will follow that the mean age at death of a group of parents of individuals of low mean age at death will tend to be lower than the mean age of a group of parents of individuals of relatively high mean age at death, merely as a result of the inheritance of duration of life, and quite regardless of whether the individual of the first group had tuberculosis, while the individual of the second group did not. *Mutatis mutandis* the same consideration will apply to a group of parents of the cancerous as compared with parents of a random sample of the non-cancerous general population. The parents of the cancerous may be expected to show a higher mean age at death than the parents of a random sample of the non-cancerous population, because the cancerous themselves die at advanced ages, and we are really comparing the ages of the parents of a group of *old* people with the parents of a group of *younger* people. Because of the correlation between parent and offspring in age at death the former will tend to have a higher average than the latter.

The kind of control group that is wanted is clearly shown if the problem itself is more precisely stated. Does the mean age at death of the parents of a group of tuberculous persons (or cancerous persons) differ sensibly from the mean age at death of the parents of a group of non-tuberculous (or non-cancerous) persons, these latter persons *having the same age distribution as the group of tuberculous (or cancerous) persons?* Putting the question in this way restricts the problem to one variable, the presence or absence of tuberculosis (or cancer) in the individual, rather than two variables, namely the presence or absence of the disease *and* the age distribution of the groups having it. One may suspect that Bauer perhaps overlooked this point in the investigation which led to his remark already quoted regarding the age at death of the parents of the cancerous.

Having put the problem in the form last stated I proceeded to get proper control groups in the following way. Frequency distributions were made of the ages of the tuberculous (or cancerous) *individuals*. Then, beginning with History 1, each non-tuberculous individual in order was taken, without reference to the age of the parents, and his or her age checked off against the appropriate class in the age distribution of the tuberculous. This proc-

[5] Beeton, M., and Pearson, K., Inheritance of the duration of life and the intensity of natural selection in man. *Biometrika*, vol. 1, pp. 50–89, 1901.

[6] Bell, A. G., *The duration of life and conditions associated with longevity. A study of the Hyde genealogy*, Washington, pp. 57, 4to, 1918.

ess was continued until a group of non-tuberculous persons was obtained *having exactly the same age distribution* as the group of tuberculous persons. Individuals were taken from different families in this way in about the same proportion that tuberculous had come from the same families, so that there might be no undue weighting of some families in the control as compared with the diseased group. These non-tuberculous and non-cancerous individuals having been picked out in the way described, the four items of information mentioned above were put upon punched cards. Then the whole material, from diseased and control individuals, was tabulated in appropriate classes, *according to the ages of the parents*. The groups of non-tuberculous and non-cancerous individuals having the same age distribution as the corresponding groups of diseased individuals are called the Selected Controls in what follows.

DATA REGARDING THE AGE OF PARENTS

I shall consider first the tuberculous groups, as the material is much more abundant than for the cancerous.

Tables 102 and 103 present the frequency distributions of age of *parents* (*not* of the individuals themselves).

It will be noted at once that we are dealing here with a respectable amount of material. There are, in all the groups taken together, 985 pairs of parents (985 fathers and 985 mothers) of tuberculous persons. The tuberculous persons themselves were, of course, of all ages from infancy up.

The means and standard deviations from the distributions of tables 102 and 103 are exhibited in tables 104 and 105. In these tables I have also placed at the end the constants derived from the Random Control groups.

For convenience in the comparison and analysis of the results it is desirable to have the evidence in two additional forms. First the differences together with their probable errors between corresponding groups of the parents of the tuberculous and the parents of the non-tuberculous (Selected Controls), in respect of means and standard deviations. These differences are represented in table 106.

In the second place graphic delineations of the frequency distributions are desirable. These are given in figures 56 to 63 inclusive.

Before discussing the results regarding tuberculosis it is desirable to present the data regarding cancer. This is done in table 107 (frequency distributions), table 108 (biometric constants) and table 109 (differences).

It will be noted that the cancer experience is much smaller than the tuberculous. There are but 131 pairs of parents of the cancerous (131 fathers and 131 mothers).

TABLE 102

Age distribution of fathers of tuberculous and of non-tuberculous persons

AGE OF FATHER	TUBERCULOUS PERSON				NON-TUBERCULOUS PERSONS (SELECTED CONTROL)			
	Dead		Living		Dead		Living	
	Father dead	Father living	Father dead	Father living	Father dead	Father living	Father dead	Father living
20–29	12	6	8		8	1	9	6
30–39	41	1	31	21	37	7	20	20
40–49	94	21	29	32	73	14	34	41
50–59	71	41	47	43	84	40	36	43
60–69	119	37	34	45	99	37	37	33
70–79	106	27	24	10	121	33	31	10
80–89	45	8	7	6	71	8	11	4
90–99	14	2		1	9	3	2	
100–109	2				2			1
Totals...	504	143	180	158	504	143	180	158

TABLE 103

Age distribution of mothers of tuberculous and of non-tuberculous persons

AGE OF MOTHER	TUBERCULOUS PERSONS				NON-TUBERCULOUS PERSONS (SELECTED CONTROLS)			
	Dead		Living		Dead		Living	
	Mother dead	Mother living	Mother dead	Mother living	Mother dead	Mother living	Mother dead	Mother living
10–19	1	1					1	
20–29	16	2	5	4	10	2	9	9
30–39	71	6	25	36	28	10	27	35
40–49	62	36	35	43	42	32	29	52
50–59	65	55	31	42	72	50	22	42
60–69	64	64	29	37	90	63	34	33
70–79	88	47	24	19	108	52	25	9
80–89	48	11	4	1	62	13	7	3
90–99	9	1	1	2	12	1		1
Totals...	424	223	154	184	424	223	154	184

TABLE 104

Constants for age of fathers of tuberculous and of non-tuberculous persons

PERSON	FATHER	TUBERCULOUS PERSONS		NON-TUBERCULOUS PERSONS (SELECTED CONTROL)		NON-TUBERCULOUS PERSONS (RANDOM CONTROL)	
		Mean age of father	Standard deviation	Mean age of father	Standard deviation	Mean age of father	Standard deviation
Dead	Dead	61.35±0.49	16.32±0.35	63.51±0.48	16.07±0.34	68.16±0.48	14.51±0.34
Living	Dead	54.33±0.75	14.95±0.53	57.17±0.81	16.05±0.57		
Dead	Living	60.73±0.77	13.72±0.55	62.55±0.74	13.13±0.52	76.02±0.49	6.76±0.35
Living	Living	55.82±0.68	12.75±0.48	53.17±0.73	13.56±0.51		

TABLE 105

Constants for age of mothers of tuberculous and of non-tuberculous persons

PERSON	MOTHER	TUBERCULOUS PERSONS		NON-TUBERCULOUS PERSONS (SELECTED CONTROL)		NON-TUBERCULOUS PERSONS (RANDOM CONTROL)	
		Mean age of mother	Standard deviation	Mean age of mother	Standard deviation	Mean age of mother	Standard deviation
Dead	Dead	58.87±0.60	18.17±0.42	64.72±0.52	15.96±0.37	65.43±0.58	16.08±0.41
Living	Dead	54.55±0.81	14.94±0.57	54.42±0.90	16.47±0.63		
Dead	Living	61.14±0.57	12.68±0.40	61.82±0.58	12.91±0.41	74.01±0.41	10.06±0.39
Living	Living	52.77±0.69	13.84±0.49	50.38±0.67	13.40±0.47		

TABLE 106

Difference between parents of tuberculous and non-tuberculous persons in mean age and standard deviation of age

PERSON	PARENT	DIFFERENCE IN MEANS	DIFFERENCE IN STANDARD DEVIATIONS
		Tbc.—Non-tbc.	Tbc.—Non-Tbc.
	Father		
Dead	Dead	−2.16±0.69	+0.25±0.49
Living	Dead	−2.84±1.10	−1.10±0.78
Dead	Living	−1.82±1.07	+0.59±0.76
Living	Living	+2.65±1.00	−0.81±0.70
	Mother		
Dead	Dead	−5.85±0.79	+2.21±0.56
Living	Dead	+0.13±1.21	−1.53±0.85
Dead	Living	−0.68±0.81	−0.23±0.57
Living	Living	+2.39±0.96	+0.44±0.68

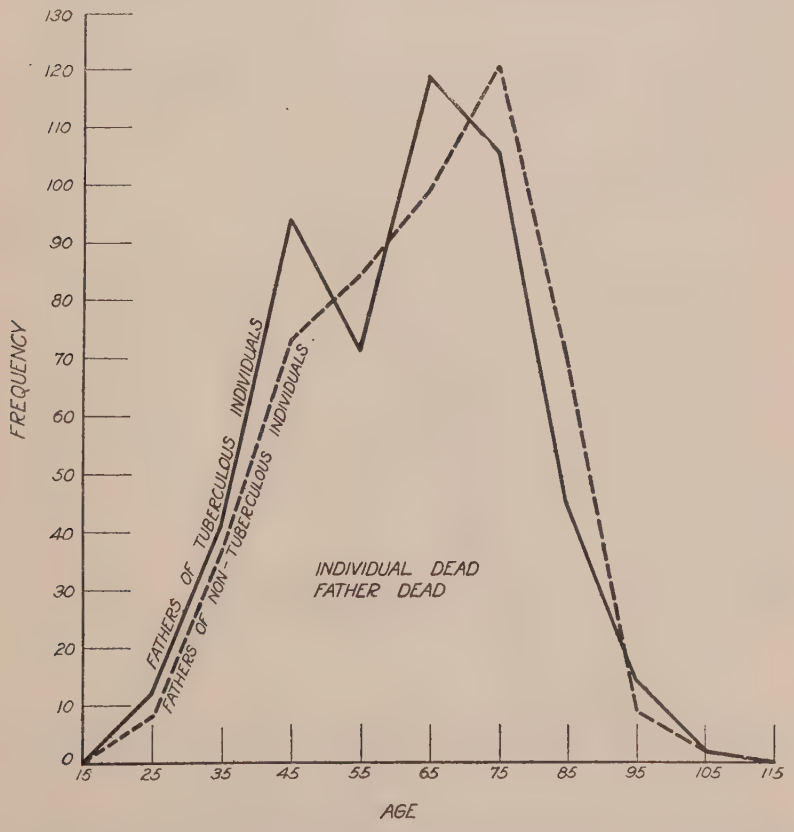

FIG. 56. FREQUENCY POLYGONS SHOWING THE AGE DISTRIBUTION OF DEAD FATHERS OF DEAD (*a*) TUBERCULOUS (SOLID LINE) AND (*b*) NON-TUBERCULOUS (BROKEN LINE) INDIVIDUALS

TABLE 107

Age distributions of parents of cancerous and of non-cancerous persons

AGE OF FATHER OR MOTHER	CANCEROUS PERSONS				NON-CANCEROUS PERSONS (SELECTED CONTROL)			
	Father dead	Father living	Mother dead	Mother living	Father dead	Father living	Mother dead	Mother living
20–29	2		2				1	
30–39	4		5		6		1	
40–49	11	1	9	2	11		5	1
50–59	19	2	17	2	17	2	24	4
60–69	31	2	22	4	14	3	28	6
70–79	35	5	25	11	37	4	29	10
80–89	13	1	19	3	29	1	18	
90–99	4		8	2	4	1	1	3
100–109	1				2			
Totals...	120	11	107	24	120	11	107	24

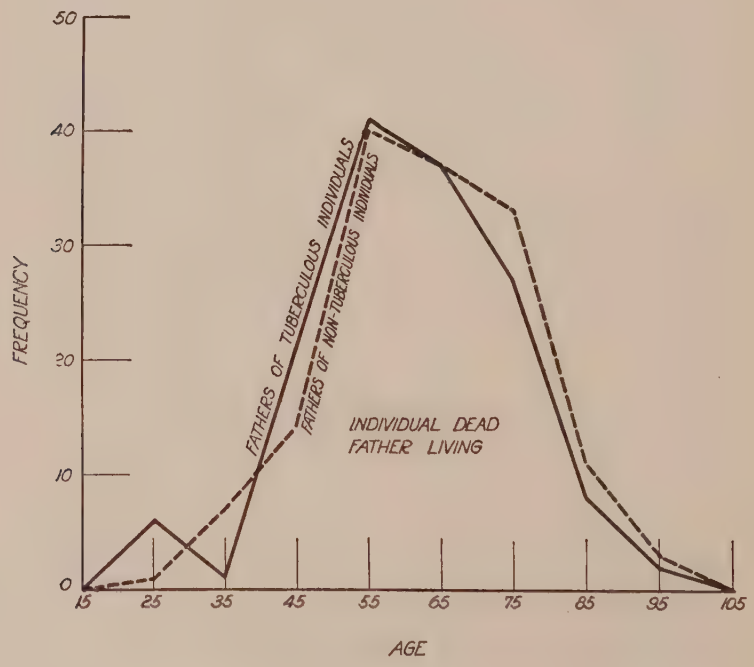

FIG. 57. LIKE FIGURE 56, BUT FOR LIVING FATHERS OF DEAD INDIVIDUALS

TABLE 108

Constants for age of parents of cancerous and of non-cancerous persons

PERSON	PARENT	CANCEROUS PERSONS		NON-CANCEROUS PERSONS (SELECTED CONTROL)		NON-CANCEROUS PERSONS (RANDOM CONTROL)	
		Mean age of parent	Standard deviation	Mean age of parent	Standard deviation	Mean age of parent	Standard deviation
	Father						
Dead	Dead	66.33±0.90	14.66±0.64	69.83±0.97	15.76±0.69	68.16±0.48	14.51±0.24
Dead	Living	67.73±2.23	10.98±1.58	71.36±2.26	11.13±1.60	76.02±0.49	6.76±0.35
	Mother						
Dead	Dead	67.71±1.07	16.36±0.75	67.52±0.83	12.69±0.59	65.43±0.58	16.08±0.41
Dead	Living	72.08±1.71	12.41±1.21	70.42±1.69	12.24±1.19	74.01±0.41	10.06±0.39

TABLE 109

Difference between parents of cancerous and non-cancerous persons in mean age and standard deviation of age

PARENT	DIFFERENCE IN MEANS	DIFFERENCE IN STANDARD DEVIATIONS
	Cancerous—Non-cancerous	Cancerous—Non-cancerous
Father		
Dead	−3.50±1.32	−1.10±0.94
Living	−3.63±3.17	−0.15±2.25
Mother		
Dead	+0.19±1.35	+3.67±0.95
Living	+1.66±2.40	+0.17±1.70

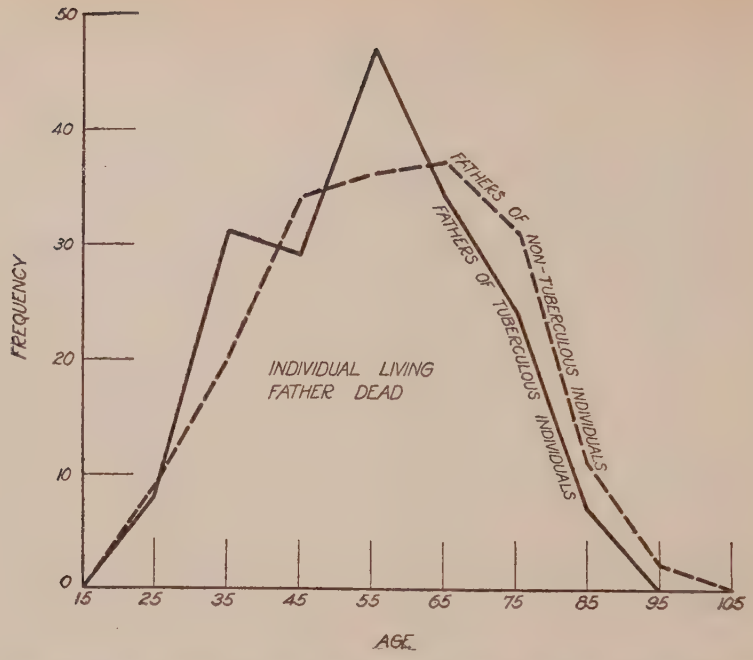

FIG. 58. LIKE FIGURE 56, BUT FOR DEAD FATHERS OF LIVING INDIVIDUALS

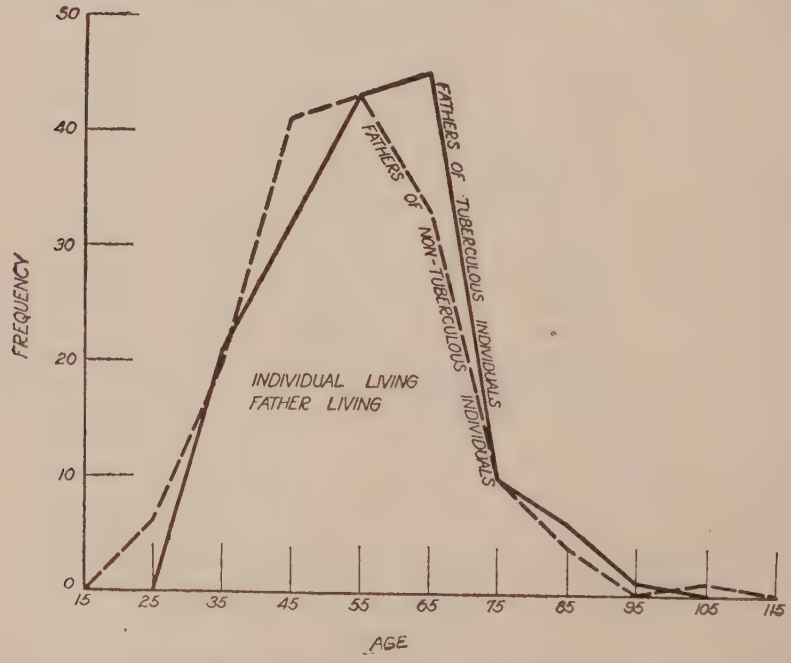

FIG. 59. LIKE FIGURE 56, BUT FOR LIVING FATHERS OF LIVING INDIVIDUALS

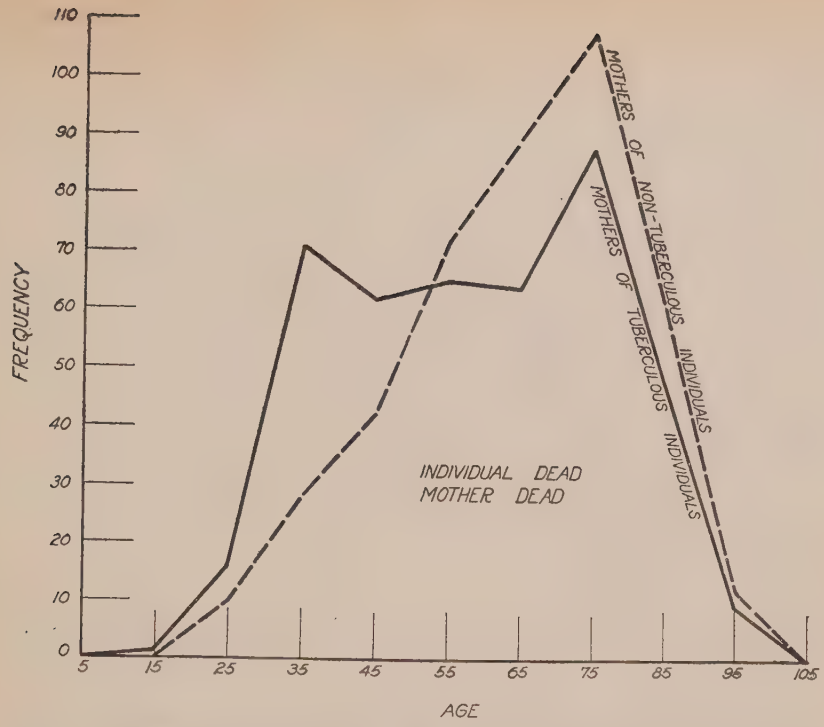

FIG. 60. FREQUENCY POLYGONS SHOWING THE AGE DISTRIBUTION OF DEAD MOTHERS OF DEAD (*a*) TUBERCULOUS (SOLID LINE) AND (*b*) NON-TUBERCULOUS (BROKEN LINE) INDIVIDUALS

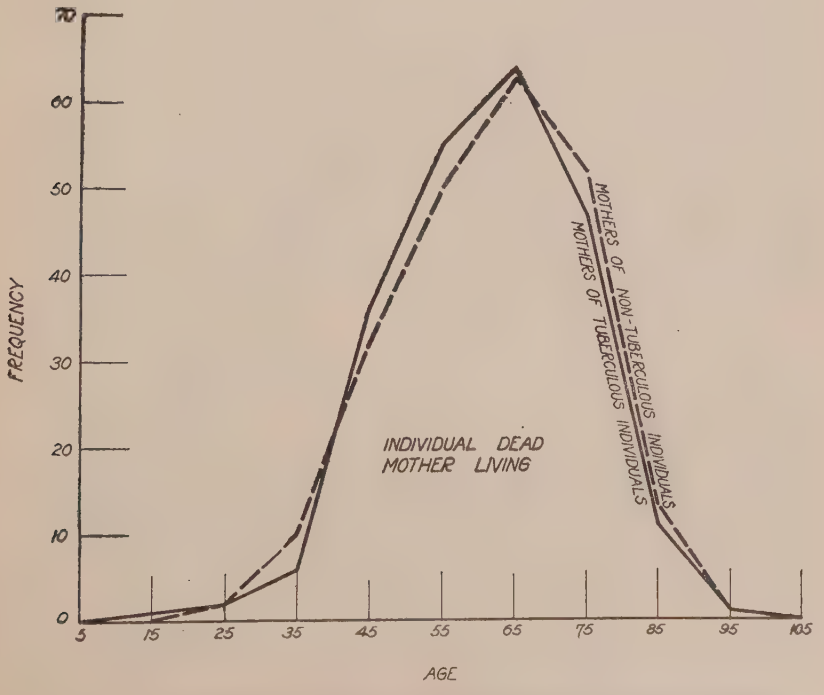

FIG. 61. LIKE FIGURE 60 BUT FOR LIVING MOTHERS OF DEAD INDIVIDUALS

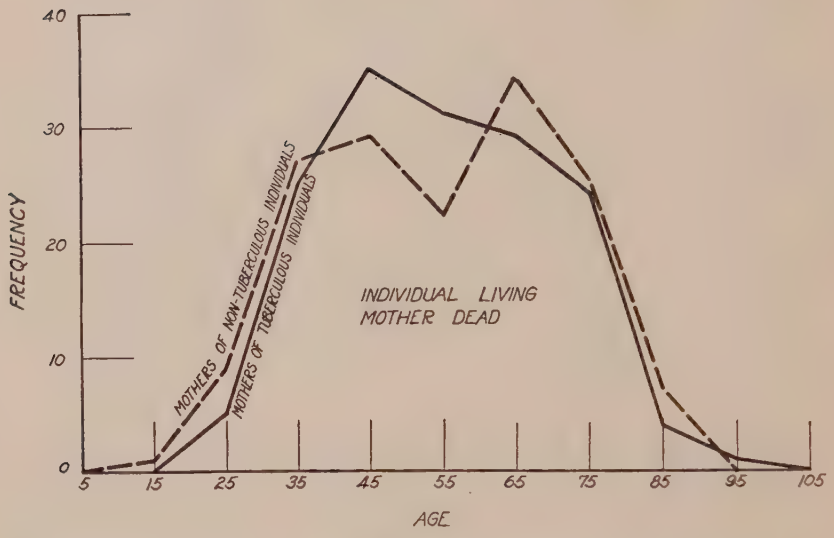

FIG. 62. LIKE FIGURE 60, BUT FOR DEAD MOTHERS OF LIVING INDIVIDUALS

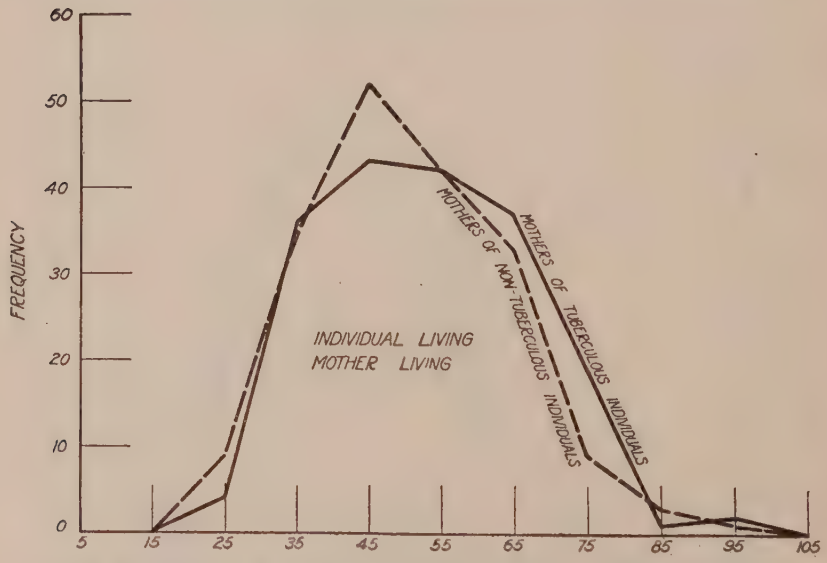

FIG. 63. LIKE FIGURE 60, BUT FOR LIVING MOTHERS OF LIVING INDIVIDUALS

DISCUSSION OF RESULTS

From the data presented we note the following points:

1. The fathers of tuberculous individuals, whether living or dead, die at an average age of between two and three years *less* than the average age at death of fathers of non-tuberculous individuals (of the same age distribution as the tuberculous). These differences are probably, but not certainly, significant statistically in each of the two groups.

2. The mothers of tuberculous individuals (themselves dead) die at an average age of nearly six years *less* than that of the dead mothers of non-tuberculous individuals (of the same age distribution as the tuberculous). The difference is certainly significant statistically, being more than 7 times its probable error. Between the dead mothers of living tuberculous and non-tuberculous individuals there is no significant difference in average age at death.

3. The average age of the living fathers and mothers of dead tuberculous individuals is in each case somewhat *less* than that of the living parents of dead non-tuberculous individuals (of the same age distribution as the tuberculous), but the differences cannot be regarded as significant, having regard to their probable errors.

4. The living mothers and fathers of living tuberculous individuals exhibit an average age of between two and three years *higher* than that of the living parents of the non-tuberculous of the same distribution.

5. Generally there are no significant differences in the variability (as measured by standard deviation) of the parents of the tuberculous and the parents of the non-tuberculous in respect of age. The only exception is in the dead mothers of the dead individuals, where the difference between standard deviations is approximately 4 times its probable error.

6. We can get a larger experience and consequently smaller probable errors by combining both sets of dead fathers (and similarly mothers) together for both dead and living individuals. This has been done with the following results:

All dead fathers of tuberculous individuals:	
Mean age at death	59.50 ± 0.42 years
Standard deviation	16.27 ± 0.30 years
All dead fathers of non-tuberculous individuals:	
Mean age at death	61.84 ± 0.42 years
Standard deviation	16.31 ± 0.30 years
Difference in mean age at death	-2.34 ± 0.59 years

This must be regarded as an almost certainly significant difference.

Proceeding in the same way with the mothers of the tuberculous and non-tuberculous we have:

All dead mothers of tuberculous individuals:	
Mean age at death	57.72 ± 0.49 years
Standard deviation	17.47 ± 0.35 years
All dead mothers of non-tuberculous individuals:	
Mean age at death	61.97 ± 0.47 years
Standard deviation	16.73 ± 0.33 years
Difference in mean age at death	-4.25 ± 0.68 years

This difference is certainly significant, and it may be safely concluded that, for completed lives (parents dead), both the mothers and fathers of tuberculous persons have a mean age at death lower by more than two years in the case of fathers and more than four years in the case of mothers than the mean age at death of the parents of non-tuberculous persons of the same age distribution as the tuberculous. These are not large amounts, but are significant in comparison with their probable errors. If duration of life may be taken as an indicator of constitutional soundness we may conclude that the parents of the tuberculous are, on the average, of somewhat weaker constitutions organically than the parents of the non-tuberculous.

7. It will be noted that the random control means, as set forth in the last columns of tables 104 and 105, enormously exaggerate the difference between the ages at death of the parents of the tuberculous and the parents of the non-tuberculous. But this exaggeration is, for reasons explained earlier, wholly fallacious. The figures are given merely to show the justice of these earlier remarks.

8. Turning to the data for the parents of the cancerous it will be noted first that, on account of the paucity of data, it has not been deemed advisable to subdivide the material into so many classes as in the case of the tuberculous. We have considered only cases in which the individual was dead of cancer.

9. The dead fathers of cancerous individuals have a mean age at death three and one-half years *lower* than the mean age at death of the dead fathers of non-cancerous individuals of the same age distribution as the cancerous. The numbers are so small, however, that this difference cannot certainly be regarded as significant, having regard to its probable error. The remarkable thing is, however, that the difference is in *the same direction* as in the case of the parents of the tuberculous and the non-tuberculous. In other words, these critical data lend no support to Bauer's (*loc. cit.*) view that the parents of the cancerous are innately of strong constitution. On

the contrary the figures indicate, for fathers at least, that just as in the case of tuberculosis the parents of the cancerous have a somewhat *lower* duration of life than the parents of those not afflicted with the disease in question.

10. The living fathers of those dead of cancer have, as a group, a mean age at death 3.63 years *lower* than the living fathers of non-cancerous persons of the same age distribution as the cancerous.

11. The mothers of the cancerous, whether living or dead, show no significant difference in mean age from the mothers of the non-cancerous, having the same age distribution as the cancerous. The female experience is, however, even smaller than the male, and it is entirely possible that with larger experience the mothers might show a result of the same sort as that exhibited by the fathers.

12. The parents of the cancerous have generally higher mean age, and by considerable amounts, whether dead or living, than the parents of the tuberculous. This is a result of the fact set forth earlier that duration of life is inherited, and cancer is a disease of advanced ages while tuberculosis is a disease of early life.

CONCLUSIONS

A study of the question of the ages at death of the parents of the tuberculous and the cancerous, on the basis of critically collected new material, shows that, for completed lives (parents dead), the mean age at death of the parents of a group of tuberculous individuals is significantly, but not greatly (two to four years), *lower* than the mean age at death of the parents of a group of non-tuberculous persons having the same age distribution as the tuberculous group. The same thing is true of the fathers of a group of cancerous individuals. The experience for mothers of the cancerous is too meagre to give definite results. In so far as duration of life may be taken statistically as an indicator of soundness of biological constitution, it follows that in some degree, though not considerably, both the tuberculous and the cancerous are of weaker stock constitutionally than those not having these diseases. Incidentally the results of this investigation furnish new evidence, of a different sort than any heretofore offered, of the inheritance of duration of life.

CHAPTER XIII

Comparing the Mortality of Man with That of Other Animals[1]

The appropriate procedure for gaining an adequate scientific description of the mortality in any aggregate of living things is well understood, and has been widely practiced for nearly three centuries by vital statisticians and actuaries. It consists fundamentally in setting up, from observations over a sufficient time, the specific death rates at ages, for a chosen time unit, which unit in the case of man is commonly taken as one year. From these age specific death rates all other actuarial functions of the mortality, such as survivorship, absolute mortality, expectation of life, etc., are derived, by mathematical procedures which are in essence simple enough, if sometimes complicated in the practical computations.

Now, while all this is well understood, when one embarks upon a comprehensive general biological investigation of the laws of mortality and duration of life, he is presently confronted with a practical difficulty. How shall he compare the mortality of two organisms whose total life spans are so widely different in extent of time that it is in practice quite impossible to measure or express them in the same unit?

In a series of papers[2] from the writer's laboratory exact data have been

[1] This chapter is based upon two papers: Pearl, R. Experimental studies on the duration of life. VI. A comparison of the laws of mortality in *Drosophila* and in man, *Amer. Nat.*, vol. 56, pp. 398–405, 1922; Pearl, R., and Doering, Carl R., A comparison of the mortality of certain lower organisms with that of man, *Science*, vol. 57, pp. 209–212, 1923.

[2] Pearl, R., and Parker, S. L., Experimental studies on the duration of life. I. Introductory discussion of the duration of life in *Drosophila*, *Amer. Nat.*, vol. 55, pp. 481–509, 1921.

Idem, II. Hereditary differences in duration of life in line-bred strains of *Drosophila*, *ibid.*, vol. 56, pp. 174–187, 1922.

Idem, III. The effect of successive etherizations on the duration of life of *Drosophila*, *ibid.*, vol. 56, pp. 273–280, 1922.

Idem, IV. Data on the influence of density of population on duration of life in *Drosophila*, *ibid.*, vol. 56, pp. 312–321, 1922.

Idem, V. On the influence of certain environmental factors on duration of life in *Drosophila*, *ibid.*, vol. 56, pp. 385–398, 1922.

Pearl, R., VI. A comparison of the laws of mortality in *Drosophila* and in man, *ibid.*, vol. 56, pp. 398–405, 1922.

Pearl, R., and Parker, S. L., New experimental data on the influence of density of population upon duration of life in *Drosophila*, *Amer. Jour. Hyg.*, vol 3, pp. 94–97, 1923.

presented regarding the mortality of the fruit fly, *Drosophila melanogaster*, under a variety of conditions. Complete life tables have been constructed for this organism. The normal wild type fly has a mean duration of life in the adult or imaginal stage of around forty to fifty days, depending upon circumstances, and an extreme upper limit of imaginal life, under any experimental conditions yet realized in our work, of from ninety to one hundred days. Thus it is apparent that *Drosophila* life in days has roughly the same quantitative aspects that human life does in years.

Upon what basis shall any life table function, say l_x, of the *Drosophila* life table be compared with that of man? The life span of one of these organisms is best measured in days, while that of the other is measured in years. This fact, however, offers no insuperable difficulty to the comparison. What is needed is to superimpose the two curves so that at least two *biologically equivalent* points coincide. The best two points would be the beginning and the end of the life span. But in the case of *Drosophila* our life tables start with the beginning of *imaginal* life only. The larval and pupal durations are omitted.

I think we can get at this starting point more exactly by putting the human and *Drosophila* l_x curves together as a starting point at the age for each organism *where the instantaneous death rate* q_x *is a minimum.* In the case of *Drosophila*, I think we are safe in concluding, on the basis of the work of Loeb and Northrop[3] as well as from our own observations, that this point is at or very near the beginning of imaginal life. We shall accordingly take *Drosophila* age one day as this point. Our life tables show that certainly *after* this time q_x never again has so low a value.

The latest edition of Glover's United States Life Tables gives for white males in the original registration states the following values for q_x: for age eleven to twelve 2.28, and for age twelve to thirteen, 2.29. We may, therefore, with sufficient accuracy take exactly twelve years as the minimum point, particularly as the l_x figures we shall have to use are tabled as of the *beginning* of the age interval.

For the other end of the life span we may conveniently take the age at which there is left but *one* survivor out of a thousand starting at age one day for *Drosophila* and age twelve years for white males. This age for wild

Pearl, R., Parker, S. L., and Gonzalez, B. M., Experimental studies on the duration of life. VII. The Mendelian inheritance of duration of life in crosses of wild type and quintuple stocks of *Drosophila melanogaster*, *Amer. Nat.*, vol. 57, pp. 153–192, 1923.

Gonzalez, B. M., VIII. The influence upon duration of life of certain mutant genes of *Drosophila melanogaster*, *ibid.*, vol. 57, pp. 289–325. 1923.

[3] *Cf. Jour. Biol. Chem.*, vol. 32, pp. 103–121, 1917, and earlier papers there cited.

TABLE 110

Survivorship of white males in original registration states on the basis of 1,000 at age 12

AGE	NUMBER ALIVE AT BEGINNING OF AGE INTERVAL l_x	AGE	NUMBER ALIVE AT BEGINNING OF AGE INTERVAL l_x	AGE	NUMBER ALIVE AT BEGINNING OF AGE INTERVAL l_x
12–13	1,000	45–46	803	78–79	194
13–14	998	46–47	792	79–80	171
14–15	995	47–48	782	80–81	150
15–16	993	48–49	771	81–82	130
16–17	990	49–50	760	82–83	110
17–18	987	50–51	749	83–84	93
18–19	983	51–52	737	84–85	77
19–20	979	52–53	725	85–86	63
20–21	975	53–54	713	86–87	51
21–22	970	54–55	699	87–88	41
22–23	965	55–56	686	88–89	32
23–24	960	56–57	671	89–90	25
24–25	955	57–58	655	90–91	19
25–26	950	58–59	639	91–92	14
26–27	944	59–60	622	92–93	10
27–28	939	60–61	604	93–94	7
28–29	934	61–62	585	94–95	5
29–30	928	62–63	566	95–96	4
30–31	922	63–64	546	96–97	2
31–32	916	64–65	525	97–98	1.59
32–33	910	65–66	504	98–99	1.01
33–34	903	66–67	482	99–100	0.63
34–35	896	67–68	459		
35–36	889	68–69	436		
36–37	881	69–70	412		
37–38	873	70–71	389		
38–39	865	71–72	364		
39–40	857	72–73	340		
40–41	849	73–74	315		
41–42	840	74–75	291		
42–43	831	75–76	266		
43–44	822	76–77	241		
44–45	812	77–78	217		

type *Drosophila* is, to the nearest whole figure, ninety-seven days. To determine it for white males we have table 110.

From this it appears that there is almost exactly one survivor at ninety-eight years. So then we have as biologically equivalent life spans

97 *days* of *Drosophila* life as imago = 86 *years* of human life

Whence it follows that

1 day of *Drosophila* life = 0.8866 year of human life

and

1 year of human life = 1.1279 days of *Drosophila* life

We have calculated a life table for a third form, in addition to *Drosophila* and man, namely, the rotifer *Proales decipiens*, on the basis of data as to its mortality recently published by Dr. Bessie Noyes.[4]

Miss Noyes provides in her paper, in two different but apparently homogeneous series, data on the life history of 1454 individuals. The observations were taken only once in twenty-four hours, an interval far too long to give a smooth curve for an animal having a maximum total life span of only about eight days. This fact makes the construction of a life table more difficult and much less accurate than if the observations had been more closely spaced. It is as though one tried to construct a life table for man from data as to age at death recorded only to the nearest decade.

TABLE 111

Observed and calculated q_x values from Noyes's data on Proales

DAYS OF LIFE	OBSERVED DEATH RATE (PER 1000) WITHIN INTERVAL	CALCULATED q_x	CALCULATED l_x (NUMBER LIVING AT BEGINNING OF EACH INTERVAL
0–0.9	0	0.06	1000.0
1.0–1.9	1.4	1.39	999.9
2.0–2.9	9.6	9.99	998.5
3.0–3.9	47.3	44.98	988.5
4.0–4.9	136.5	144.60	944.0
5.0–5.9	393.9	349.90	807.5
6.0–6.9	575.9	653.50	525.0
7.0–7.9	1000.0	956.10	181.9
8.0–8.9			80.0
9.0–9.9			0

Taking the data as they stand, however, the central death-rates were computed and graduated with the results shown in table 111.

The q_x values were graduated by the following expression (with origin at 0 age, or birth) which is of a type found useful in previous work with *Drosophila* life curves:

$$\log q_x = -1.0783 + 0.7041x - 0.0452x^2 + 1.5080 \log x$$

[4] Noyes, B., Experimental studies on the life-history of a rotifer reproducing parthenogenetically (*Proales decipiens*), *Jour. Exp. Zool.*, vol. 35, pp. 225–255, 1922.

TABLE 112

Survivorship distributions (l_x) for each centile of the comparable life spans of (a) wild type Drosophila males, (b) white men in the original registration states in 1910, and (c) Proales decipiens

CENTILE OF COMPARABLE LIFE SPAN	NUMBERS ALIVE AT BEGINNING OF CENTILE AGE INTERVAL			CENTILE OF COMPARABLE LIFE SPAN	NUMBERS ALIVE AT BEGINNING OF CENTILE AGE INTERVAL		
	Drosophila	Man	*Proales*		*Drosophila*	Man	*Proales*
0–1	1000	1000	1000	38–39	571	806	984
1–2	991	998	1000	39–40	555	797	982
2–3	981	996	1000	40–41	540	788	979
3–4	972	994	1000	41–42	524	779	976
4–5	963	991	1000	42–43	508	770	973
5–6	954	989	1000	43–44	492	760	969
6–7	945	986	1000	44–45	475	750	965
7–8	935	983	1000	45–46	459	740	960
8–9	926	980	1000	46–47	443	730	955
9–10	917	976	1000	47–48	426	720	950
10–11	907	972	1000	48–49	410	709	944
11–12	898	968	1000	49–50	393	697	937
12–13	888	963	1000	50–51	377	685	930
13–14	879	959	1000	51–52	360	673	922
14–15	869	955	1000	52–53	344	659	914
15–16	859	950	1000	53–54	328	645	905
16–17	849	946	1000	54–55	312	631	895
17–18	839	941	1000	55–56	296	616	884
18–19	828	936	999	56–57	280	601	872
19–20	818	932	999	57–58	265	585	859
20–21	807	927	999	58–59	250	568	845
21–22	796	922	999	59–60	235	551	830
22–23	785	916	999	60–61	221	533	815
23–24	773	911	999	61–62	207	515	799
24–25	761	905	998	62–63	193	496	782
25–26	749	899	998	63–64	180	477	763
26–27	737	893	997	64–65	167	458	743
27–28	725	887	997	65–66	155	438	723
28–29	712	880	996	66–67	143	418	701
29–30	699	874	996	67–68	132	397	678
30–31	686	867	995	68–69	121	377	655
31–32	672	860	994	69–70	111	356	630
32–33	659	852	993	70–71	102	335	605
33–34	645	845	992	71–72	92	314	579
34–35	630	838	991	72–73	84	292	552
35–36	616	830	990	73–74	76	271	525
36–37	601	822	988	74–75	68	250	497
37–38	586	814	986	75–76	61	229	469

TABLE 112—*Continued*

CENTILE OF COMPARABLE LIFE SPAN	NUMBERS ALIVE AT BEGINNING OF CENTILE AGE INTERVAL			CENTILE OF COMPARABLE LIFE SPAN	NUMBERS ALIVE AT BEGINNING OF CENTILE AGE INTERVAL		
	Drosophila	Man	*Proales*		*Drosophila*	Man	*Proales*
76–77	55	208	440	89–90	9	28	109
77–78	49	189	411	90–91	7	22	91
78–79	43	169	382	91–92	6	17	75
79–80	38	151	354	92–93	5	13	61
80–81	34	133	326	93–94	4	10	49
81–82	30	117	298	94–95	3.43	8	38
82–83	26	101	270	95–96	2.80	6	28
83–84	22	87	244	96–97	2.27	4	20
84–85	19	74	218	97–98	1.84	3	13
85–86	17	62	194	98–99	1.47	2.11	8
86–87	14	52	171	99–100	1.18	1.37	4
87–88	12	43	149	100	0.94	0.87	1
88–89	10	35	128				

It is evident from the data of table 111 that the fit is reasonably good, probably as good as one could expect with observations so rough in respect of age at death.

We are now in position to make an exact comparison between the life tables of the three organisms. This may be done perhaps most instructively by setting up l_x lines for the three forms on the basis of age in *centiles of the life span*, rather than days or years. That is to say, the whole comparable life spans (as here defined) of ninety-seven days in *Drosophila* and of eighty-six years for white males and of just under nine days in *Proales* were each divided into 100 equal parts, and the survivors at the attainment of the beginning of each centile interval were then computed. The results, so far as concerns the survivorship function l_x, are shown in table 112.

In order that it may be seen how the forces of mortality operate in *Proales* as compared with man and *Drosophila*, the diagram shown as figure 64 is presented.

Comparing the three curves, we note the following points:

1. The *Proales* curve lies above the other two at all parts of the comparable life span. This means that out of 1000 individuals starting together at biologically equivalent points in the life span (*i.e.*, at the age when q_x is a minimum for each organism) at any subsequent age centile there will be more surviving rotifers than men, and more surviving men than flies.

2. The median durations of life, or, put in another way, the ages prior to which just 500 of the 1000 individuals starting together will have died, are approximately:

For *Proales*	74	per cent of the equivalent life span
For man	62	per cent of the equivalent life span
For *Drosophila*	42.5	per cent of the equivalent life span

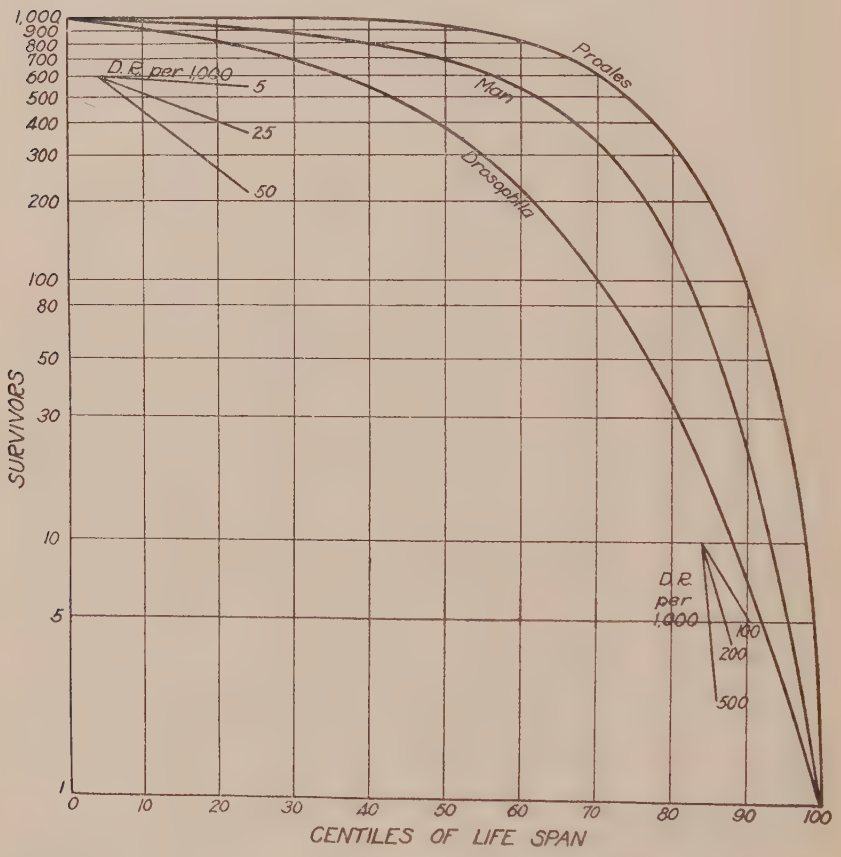

FIG. 64. SHOWING SURVIVORSHIP DISTRIBUTIONS FOR (*a*) ROTIFER *Proales Decipiens*, (*b*) MAN (MALES IN ORIGINAL REGISTRATION STATES, 1910), AND (*c*) *Drosophila Melanogaster* (WILD TYPE MALES)

The death rates corresponding to given slopes of the l_x line are also given by the groups of fine lines at the two ends of the diagram.

3. The comparison the other way about indicates that when 50 per cent of the equivalent life spans have been passed there are still surviving:

In *Proales*	93.0 per cent of the individuals starting
In man	68.5 per cent of the individuals starting
In *Drosophila*	37.7 per cent of the individuals starting

The outstanding thing about the life curve for *Proales* from Miss Noyes's figures is that it approaches nearer to the theoretically possible right-angled form, in which all the individuals live to a given age x and then all die at once, than any other that has yet been observed. Whether this is the result of (*a*) the greater uniformity of environment, on the average, for the *Proales* under the experimental conditions than for the other forms, or (*b*) the greater uniformity of the population in genetic constitution, consequent upon the fact that *Proales* reproduces parthenogenetically and that all of Miss Noyes's cultures were descended from at most not over six different individuals, or (*c*) a combination of both, can not be definitely stated. Both of the factors mentioned undoubtedly do in some degree operate to produce the form of life curve exhibited.

There is need for data regarding the mortality of other organisms. It is an interesting commentary on the development of biology that the distribution of mortality in respect of age is known for only three species of animal life with sufficient accuracy to permit the formation of age-specific death rates, and hence of a life table. Into every discussion of the problem of evolution, and into every attempt to determine its causes, there must necessarily enter the question of the mortality of the forms being dealt with. There seems no good reason for indefinitely continuing to handle the matter by the current methods, which are either to make large *a priori* guesses about the distribution of mortality in the particular case, or to assume that it is the same as that of man. In the nearly universal neglect of the problem of mortality and duration of life, biologists have missed an interesting and obviously important field.

PART III
PUBLIC HEALTH AND EPIDEMIOLOGY

CHAPTER XIV

National Food Consumption[1]

THE PLAN

The basis of any adequate survey of food resources must be essentially physiological, rather than one of commodities or trade. Broadly speaking the ultimate sources of food are the soil and the sun. The energy derived from the sun through the mechanism of the green plant builds up the inorganic chemical elements of the soil, air, and water into compounds which can be utilized as food by man, either directly or secondarily in the form of the products of animals which have been nourished on the primary foods of the plant world.

For the purpose of statistical analysis all nutritive materials produced and consumed fall into one or another of the following categories, which are obviously based on the considerations set forth in the preceding paragraph.

I. Primary Foods. All plant materials used as human food or fractions of such materials, and all animals or animal products in which the animal gets its nourishment from some source other than the primary feeds and fodders as defined below, consumed by man either

a. Directly as harvested, with only such sophistication as comes from cooking, such as, for example, potatoes, fish, oysters.

b. In derivative form, where by process of manufacture a food product is prepared from a raw plant product; such as, for example, wheat flour or cottonseed oil.

II. Primary feeds or fodders. All plant materials or fractions of such materials used for the nourishment of domestic animals, consumed by such animals either

a. Directly as harvested, such as the coarse grains, or

b. In derivative or manufactured form, such as manufactured feeds.

III. Secondary foods. All edible products of animals used for human food which are nourished with primary feeds and fodders, including such foods as are produced

a Directly, without involving the death of the producing animal, such as, for example, honey, eggs, or milk, and

b. Derivatively, involving the death of the animal, such as, for example, the meats.

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[1] This chapter is based upon a paper entitled "The relative contribution of the staple commodities to the national food consumption," published in *Proc. Amer. Philosoph. Soc.*, vol. 58, pp. 182–222, 1919. The same problem is treated in great detail, and all the foundational data on which the present results are based will be found in my former book, *The Nation's Food. A Statistical Study of a Physiological and Social Problem*, Philadelphia (W. B. Saunders), 1920. That book, however, is now out of print and will not be reissued. It therefore seems desirable to make some of its chief results available in this volume.

The basic idea of this classification is, of course, to allocate the nutrient resources of the nation according to the usage made of them. We have certain products of the soil, and of the seas and fresh water lakes and streams, which are directly produced and directly consumed as human food. To produce a crop of potatoes or of cod fish or oysters it is not necessary to feed out to the growing crop some other crop such as hay or grain. Therefore these are direct, primary food products. On the other hand there are many foods such as the meats, eggs, etc., where to obtain a pound of protein, or fat, or carbohydrate for human consumption it is necessary to use a certain amount of other protein, fat, and carbohydrate primarily produced as fodder or feed. Human food produced in this manner is obviously secondarily produced and cannot be allowed to count in the net nutritive balance sheet on the same basis as the primarily produced food. It is a relatively more expensive form of nourishment.

It is evident that under this classification many raw food materials will of necessity fall in part into two or more categories. For example, to take the case of wheat, the major part of the raw grain is ground into flour and as such used as human food, but in the process of making the flour there is produced a certain amount of feeding stuffs, bran, middlings, etc., which only indirectly contribute to human nutrition through the products of animals which eat these wheat feeds. Finally a certain small proportion of the wheat grain is fed directly as such to livestock. Similar considerations apply to very many other food materials. That all this adds a considerable complexity to the problem is evident. But it is equally clear that if anything approaching reliability in the final result is to be attained, due regard must be paid to these complicated subdivisions in usage of the raw food materials. Otherwise the same nutritive material will be duplicated in the accounting and a misleading result reached.

The general plan of this study was first to determine as accurately as possible from existing official statistics for each year from 1911–1912 to 1917–1918, inclusive, the amount of the basic nutrients, protein, fat and carbohydrate: (*a*) produced, (*b*) imported, (*c*) exported, classifying the results under the main headings given above. From this tabulation as a base one may then proceed to calculations of consumption.

In all cases where investigation showed it to be necessary deductions were made for the following kinds of reasons:

a. Loss of commodity in storage
b. Spoilage of commodity in storage
c. Loss of commodity in transit
d. Spoilage of commodity in transit

e. Loss by vermin
f. Amount fed to livestock
g. Amount used for technical, non-food purposes, including the manufacture of alcoholic beverages
h. Inedible refuse

The effort was made, in the most careful and critical manner possible, to have the final figures for human food consumption represent *net* values. It is believed that this desideratum has been substantially attained.

In making up the basic tables each commodity or derivative of a commodity was listed separately and converted as such into nutrient values. In the matter of units of measure the following general plan was followed: In all basic tables the quantities of production, export and import are first given in the American units (bushels, pounds, gallons, etc.) of the original statistics. These quantities were then all converted into metric tons.[2] All nutrient values, protein, fat, and carbohydrate are given in metric tons. Energy values are expressed in millions of small calories.[3]

Regarding the sources of the basic statistics the following general statement may be made here.[4] For *production* figures the fundamental sources, in the case of primary products, are the successive *Year Books* of the United States Department of Agriculture. Each volume of this publication carries as an appendix statistical tables giving the Department's official figures of crop production. A secondary source for crop production figures was found in the successive volumes of the *Monthly Crop Report* of the United States Department of Agriculture. Its figures are again official and form the basis of the tabulation of the *Year Book*, but frequently give more detailed information. Reliable statistics of the derivative products such as flour, meals, etc., are much more difficult to obtain than crop production figures, for the reason that they are not officially collected and published. In this field resort was had to a variety of sources, such as trade papers, census returns, special *ad hoc* inquiries of manufacturers, etc.

Export and import figures were taken from the official reports (annual and monthly) of the foreign commerce of the United States compiled by the Department of Commerce. In a few cases where it was clear from information available to the Food Administration that the official figures of the Department of Commerce were in error we have not hesitated to use other and, we believe, more correct statistics.

[2] The metric ton = 2204.6 pounds.

[3] A small calory is the amount of heat necessary to raise 1 gram of water 1°C.

[4] This statement is supplemented by more detailed source references in *The Nation's Food*.

In the computation of nutrient values use was made chiefly of the factors given by Atwater and Bryant.[5] It was necessary, in some cases, to supplement their tables from data given by Leach[6] and Henry and Morrison.[7]

All calculations in this work were repeatedly checked and every possible precaution taken to guard against error. It is too much to hope that so extensive a piece of statistical work should be without errors, but I hope that their number is small and their net significance in the final results negligible.

THE CONSUMPTION OF HUMAN FOOD IN THE UNITED STATES

There have been available only the roughest guesses as to the total domestic consumption of all but a few items of food, such as wheat and sugar. If anyone were confronted with the naïve and simple question, "How much corn, or oats, or molasses, or fish, or milk, or nuts," or any one of a long series of other foods, "is consumed annually in the United States as human food," no accurate answer could be given. Yet the question is obviously a fair one, and one which somebody in the nation ought to be able to answer with a considerable degree of accuracy. For some twenty-odd great staple commodities or groups of like commodities we are now in a position to present figures of a relatively high degree of accuracy as to consumption. On the basis of these figures it is possible to discuss effectively many interesting and important problems; such as, for example, that of the relative importance of great groups of staples, like the grains and the vegetables, in the nutrition of the people of the nation. We can calculate with accuracy the total national food bill, and so forth.

The final net results as to consumption of human foods in the United States during the seven years are set forth in table 113. In that table the results are given for the several nutrient values, protein, fat, carbohydrate and calories, only. This is the most scientific, and as soon as one becomes accustomed to it, by far the most useful way of thinking about food consumption.

The data of table 113 are summarized by years in table 114, and are shown graphically in relative form in figure 65.

[5] Atwater, W. O., and Bryant, A. P., The chemical composition of American food materials (corrected April 14, 1906), *U. S. Dept. Agr. Office of Expt. Sta. Bulletin 28* (revised edition), 1906.

[6] Leach, A. E., *Food Inspection and Analysis*, third edition, revised and enlarged by A. L. Winton, New York, 1913.

[7] Henry, W. A., and Morrison, F. B., *Feeds and Feeding*, sixteenth edition, Madison, 1916.

The first thing which impresses one about the consumption figures is their extreme uniformity from year to year, as compared with production, exports and imports. This is exactly what would be expected, of course. No matter how much production, exports and imports may fluctuate, within wide limits, the people of this country eat about the same amount each year. To have the statistical calculation come out to this result so beautifully is strong evidence of the correctness of the long and tedious preliminary calculations. There has been a rather steady small increase in total gross food consumption, but this has been very closely proportional to the increase in the population.

In the seven-year period here discussed the greatest relative advance in consumption was in respect of fat, and the least relative advance in respect of protein. Carbohydrate content and calories increased in the seven years in amount consumed to a degree intermediate between fat and protein. The protein relative line falls below the population relative line each year after 1913–1914. This means that since 1913–1914 somewhat less protein has been consumed in gross in proportion to the population. The relative line for fat was below the population line till 1914–1915, and thereafter followed it closely.

The relative figures from which figure 65 is plotted are given in table 115.

With such gratifying assurance of the smoothness of the consumption results we may proceed to an analytical discussion of the numerous highly interesting problems which center about human food consumption, and for which data have hitherto been lacking.

The first of such problems to which attention may be turned is: To what relative degree do primary, as distinguished from secondary, human foods contribute to the total nutritional intake of our population? From table 114 it is seen that of the protein consumed 47 per cent comes from primary sources and 53 per cent from secondary sources. Thus, broadly speaking, the American people get over one half of their protein from animal sources exclusive of fish, which are included in the primary foods. This fact indicates at once the importance of maintaining our animal herds intact and keeping the price of animal products at not too high a level, unless we are prepared to face the alternative of a radical and fundamental alteration in the established dietary habits of the people.

In general there has been but little change in this protein-source dietary habit in the seven years included in this study. What change there has been is in the direction of a smaller proportion of protein from secondary sources and a larger from primary, but the movement has been but slight. As would be expected, a much larger proportion of the total fat consumed in human food comes from secondary sources than is the case with protein.

TABLE 113

The consumption of human foods in the United States, 1911 to 1918 (metric tons)

COMMODITY	1911–12				1912–13			
	Protein in metric tons	Fat in metric tons	Carbohydrate in metric tons	Calories in millions	Protein in metric tons	Fat in metric tons	Carbohydrate in metric tons	Calories in millions
Grains and derivative products:								
Wheat and products	1,000,730	87,132	6,589,209	31,933,764	995,249	86,726	6,553,141	31,759,774
Corn products	205,809	99,607	1,831,949	9,283,484	203,748	98,245	1,814,153	9,189,386
Rye products	10,215	1,351	118,242	539,899	10,701	1,416	123,843	565,476
Rice and products	13,472	338	133,036	605,503	15,840	397	156,420	711,928
Other cereals	26,728	10,457	150,341	824,114	26,114	10,293	145,068	798,429
Sub-total—Grains	1,256,954	198,885	8,822,777	43,186,764	1,251,652	197,077	8,792,625	43,024,993
Vegetables:								
Legumes	66,717	4,619	174,915	1,034,622	70,279	4,830	184,215	1,089,145
Potatoes	100,861	5,604	823,688	3,843,272	136,412	7,578	1,114,025	5,197,962
Other vegetables	28,662	9,607	282,235	1,367,003	29,537	9,682	285,777	1,385,885
Sub-total—Vegetables	196,240	19,830	1,280,838	6,244,897	236,228	22,090	1,584,017	7,672,992
Sugars	454	0	3,906,511	16,021,424	455	0	4,104,958	16,835,176
Fruits:								
Apples	8,646	8,500	309,136	1,388,209	9,519	9,406	341,068	1,531,633
Oranges	1,571	392	30,223	132,398	1,589	397	30,558	133,866
Bananas	7,575	3,788	121,214	563,684	7,219	3,609	115,509	537,156
Other fruits	6,492	7,173	151,684	710,188	8,171	7,288	184,418	851,392
Sub-total—Fruits	24,284	19,853	612,257	2,794,479	26,498	20,700	671,553	3,054,047
Vegetable oils and nuts:								
Nuts	40,460	77,555	30,491	1,012,435	43,369	81,228	32,932	1,068,492
Vegetable oils	0	464,403	0	4,321,063	0	458,136	0	4,262,661
Chocolate and cocoa	8,312	19,470	16,242	280,323	7,978	18,676	15,593	268,987
Sub-total—Oils and nuts	48,772	561,428	46,733	5,613,821	51,347	558,040	48,525	5,600,140

Fish	86,948	19,176	20	552,810	85,016	18,477	20	538,436
Sub-total—All primary	1,613,652	819,172	14,669,136	74,414,195	1,651,196	816,384	15,201,698	76,725,784
Meats and meat products:								
Beef and products	546,104	516,545	1,725	7,072,130	521,798	496,439	1,649	6,784,205
Pork and products	388,745	1,963,696	2,664	19,874,512	381,901	1,926,270	2,597	19,498,083
Mutton and products	45,782	61,465	521	762,158	48,677	70,612	542	859,246
Sub-total—Meats	979,981	2,541,621	4,891	27,705,272	951,570	2,494,770	4,766	27,151,638
Poultry and eggs	235,699	165,906	0	2,508,307	239,584	168,659	0	2,549,776
Oleomargarine	680	47,038	0	440,412	774	53,539	0	501,291
Dairy products	726,604	1,368,995	880,525	19,337,072	720,632	1,358,126	872,694	19,179,262
Sub-total—All secondary	1,942,964	4,123,560	885,416	49,991,063	1,912,560	4,075,094	877,460	49,381,967
Grand total	3,556,616	4,942,732	15,554,552	124,405,258	3,563,756	4,891,478	16,079,158	126,107,751

TABLE 113—*Continued*

COMMODITY	1913–14				1914–15			
	Protein in metric tons	Fat in metric tons	Carbohydrate in metric tons	Calories in millions	Protein in metric tons	Fat in metric tons	Carbohydrate in metric tons	Calories in millions
Grains and derivative products:								
Wheat and products	1,166,243	101,745	7,679,047	37,217 595	978,806	85,316	6,446,223	31,240,761
Corn products	202,368	97,263	1,802,353	9,126,239	201,503	96,571	1,795,051	9,086,301
Rye products	11,173	1,479	129,304	590,413	11,228	1,486	129,936	593,297
Rice and products	18,636	465	184,022	837,569	13,427	337	132,597	603,495
Other cereals	29,981	12,119	158.441	885,682	28,572	11,601	149,850	840,310
Sub-total—Grains	1,428,401	213,071	9 953,167	48,657,498	1,233,536	195,311	8,653,657	42,364,164
Vegetables:								
Legumes	76,757	5,282	202,149	1,193,370	68,833	4,869	179,423	1,063,900
Potatoes	108,850	6,046	888,931	4,147,685	132,339	7,353	1,080,780	5,042,836
Other vegetables	28,605	9,178	286,216	1,379,545	33,441	10,126	304,815	1,484,680
Sub-total—Vegetables	214,212	20,506	1,377,296	6,720,600	234,613	22,348	1,565,018	7,591,416
Sugars	455	0	4,423,200	18,140,160	455	0	4,319,726	17,715,852
Fruits:								
Apples	5,792	5,701	207,210	930,502	10,256	10,141	367,567	1,650,637
Oranges	1,526	381	29,358	128,607	1,499	374	28,839	126,340
Bananas	8,222	4,111	131,567	611,836	6,914	3,447	110,304	512,951
Other fruits	6,656	7,608	150,045	708,077	7,878	6,607	169,753	783,643
Sub-total—Fruits	22,196	17,801	518,180	2,379,022	26,547	20,569	676,463	3,073,571
Vegetable oils and nuts:								
Nuts	50,244	95,092	38,381	1,247,995	50,929	95,096	38,400	1,250,900
Vegetable oils	0	541,477	0	5,038,094	0	595,041	0	5,536,504
Chocolate and cocoa	10,200	23,885	19,931	343,932	9,195	21,574	17,954	310,281
Sub-total—Oils and nuts	60,444	660,454	58,312	6,630,021	60,124	711,711	56,354	7,097,685

Fish	86,108	18,563	18	543,899	86,796	18,732	19	548,479
Sub-total—All primary	1,811,816	930,395	16,330,173	83,071,200	1,642,071	968,671	15,271,237	78,391,167
Meats and meat products:								
Beef and products	507,758	486,631	1,583	6,634,517	508,717	490,365	1,662	6,673,341
Pork and products	364,500	1,855,128	2,482	18,764,355	416,899	2,157,916	2,873	21,797,474
Mutton and products	48,684	77,397	535	922,389	42,256	64,254	462	773,384
Sub-total—Meats	920,379	2,419,465	4,586	26,321,773	967,165	2,707,408	4,980	29,193,531
Poultry and eggs	244,965	172,484	0	2,607,319	249,006	175,349	0	2,650,480
Oleomargarine	770	53,261	0	498,672	765	52,917	0	495,467
Dairy products	731,613	1,379,896	884,160	19,473,916	742,130	1,400,492	903,312	19,787,471
Sub-total—All secondary	1,897,727	4,025,106	888,746	48,901,680	1,959,066	4,336,166	908,292	52,126,949
Grand total	3,709,543	4,955,501	17,218,919	131,972,880	3,601,137	5,304,837	16,179,529	130,518,116

TABLE 113—*Concluded*

COMMODITY	1915–16				1916–17				1917–18			
	Protein in metric tons	Fat in metric tons	Carbohydrate in metric tons	Calories in millions	Protein in metric tons	Fat in metric tons	Carbohydrate in metric tons	Calories in millions	Protein in metric tons	Fat in metric tons	Carbohydrate in metric tons	Calories in millions
Grains and derivative products:												
Wheat and products	1,159,286	101,146	7,636,118	37,007,387	1,026,976	89,506	6,765,024	32,784,510	940,543	81,835	6,195,182	30,021,979
Corn products	201,163	96,173	1,792,381	9,070,259	201,709	96,269	1,797,497	9,094,401	242,395	118,845	2,155,310	10,938,521
Rye products	11,480	1,519	132,856	606,633	12,245	1,621	141,725	647,129	24,597	3,256	284,668	1,299,820
Rice and products	16,677	417	164,691	749,578	25,331	634	250,140	1,138,508	30,725	767	303,428	1,381,039
Other cereals	32,131	13,292	161,941	920,265	39,298	16,587	189,036	1,091,501	65,088	23,104	352,857	1,927,964
Sub-total—Grains	1,420,737	212,547	9,887,987	48,354,122	1,305,559	204,617	9,143,422	44,756,049	1,303,348	227,807	9,291,445	45,569,323
Vegetables:												
Legumes	59,707	4,145	153,757	913,944	75,821	5,200	198,157	1,172,609	105,578	7,325	277,203	1,638,716
Potatoes	115,422	6,412	942,629	4,398,237	93,703	5,205	765,232	3,570,508	143,167	7,953	1,169,204	5,455,418
Other vegetables	32,292	10,998	346,643	1,660,414	28,284	10,021	317,522	1,514,539	36,668	12,586	398,275	1,904,998
Sub-total—Vegetables	207,321	21,555	1,443,029	6,972,595	197,808	20,426	1,280,911	6,257,656	285,413	27,864	1,844,682	8,999,132
Sugars	455	0	4,047,276	16,598,665	455	0	4,356,901	17,868,295	439	0	4,374,194	17,939,129
Fruits:												
Apples	9,610	9,567	345,335	1,550,836	8,490	8,461	305,219	1,370,683	7,458	7,451	268,425	1,205,454
Oranges	1,523	379	29,286	128,293	2,176	544	41,912	183,597	1,109	278	21,360	93,569
Bananas	6,169	3,085	98,717	459,065	5,778	2,889	92,457	429,960	5,771	2,885	92,328	429,360
Other fruits	9,295	9,174	206,025	961,649	7,227	8,429	167,517	790,038	9,283	6,767	219,237	994,221
Sub-total—Fruits	26,597	22,205	679,363	3,099,843	23,671	20,323	607,105	2,774,278	23,621	17,381	601,350	2,722,604
Vegetable oils and nuts:												
Nuts	47,957	99,492	36,507	1,271,900	47,957	105,623	36,717	1,329,746	81,939	179,337	63,054	2,262,988
Vegetable oils	0	424,858	0	3,953,026	0	547,294	0	5,092,191	0	554,851	0	5,162,528
Chocolate and cocoa	11,880	27,881	23,200	400,975	18,554	43,530	36,235	626,117	20,083	47,273	39,177	678,641
Sub-totals—Oils and nuts	59,837	552,231	59,707	5,625,901	66,511	696,447	72,952	7,048,054	102,022	781,461	102,231	8,104,157

Fish	79,968	16,045	20	495,706	84,275	17,582	23	527,725	85,021	17,866	25	533,419
Sub-total—All primary	1,794,915	824,583	16,117,382	81,146,832	1,678,279	959,395	15,461,314	79,232,057	1,799,864	1,072,379	16,213,927	83,867,764
Meats and meat products:												
Beef and products	525,129	502,065	1,740	6,850,539	562,748	538,151	1,881	7,342,374	539,703	513,596	1,577	7,017,398
Pork and products	405,161	2,143,483	2,929	21,614,254	398,781	2,098,923	2,895	21,173,213	378,799	2,045,653	2,859	20,594,616
Mutton and products	40,286	62,637	439	750,130	36,589	56,626	406	678,884	28,298	46,853	315	553,498
Sub-total—Meats	969,077	2,700,434	5,076	29,136,535	996,527	2,685,983	5,146	29,115,663	945,277	2,602,187	4,717	28,122,722
Poultry and eggs	252,314	177,696	0	2,685,822	255,499	179,999	0	2,720,161	248,772	175,220	0	2,648,262
Oleomargarine	800	55,375	0	518,470	1,238	85,658	0	802,005	1,808	125,024	0	1,170,593
Dairy products	764,377	1,445,669	919,595	20,366,131	783,350	1,482,331	937,858	20,860,208	788,969	1,505,129	917,169	21,010,397
Sub-total—All secondary	1,986,568	4,379,174	924,671	52,706,958	2,036,614	4,433,971	943,004	53,498,037	1,984,826	4,407,560	921,886	52,951,974
Grand total	3,781,483	5,203,757	17,042,053	133,853,790	3,714,893	5,393,366	16,404,318	132,730,094	3,784,690	5,479,939	17,135,813	136,819,728

TABLE 114

Summary of consumption of human foods, primary and secondary (metric tons)

	PROTEIN	PER CENT FROM		FAT	PER CENT FROM		CARBOHYDRATE	PER CENT FROM		CALORIES (MILLIONS)	PER CENT FROM	
		Primary	Secondary		Primary	Secondary		Primary	Secondary		Primary	Secondary
1911–12	3,556,616	45	55	4,942,732	17	83	15,554,552	94	6	124,405,258	60	40
1912–13	3,563,756	46	54	4,891,478	17	83	16,079,158	95	5	126,107,751	61	39
1913–14	3,709,543	49	51	4,955,501	19	81	17,218,919	95	5	131,972,880	63	37
1914–15	3,601,137	46	54	5,304,837	18	82	16,179,529	94	6	130,518,116	60	40
1915–16	3,781,483	47	53	5,203,757	16	84	17,042,053	95	5	133,853,790	61	39
1916–17	3,714,893	45	55	5,393,366	18	82	16,404,318	94	6	132,730,094	60	40
1917–18	3,784,690	48	52	5,479,939	20	80	17,135,813	95	5	136,819,738	61	39
Total for 7 years	25,712,118	47	53	36,171,610	18	82	115,614,342	95	5	916,407,627	61	39
Average, whole period	3,673,160	47	53	5,167,373	18	82	16,516,335	95	5	130,915,375	61	39
Average, 1911–12 to 1916–17	3,654,571	46	54	5,115,279	17	83	16,413,088	95	5	129,931,315	61	39

The figures are 82 per cent from secondary sources and 18 per cent from primary. Again there has been little change in the seven years. In spite

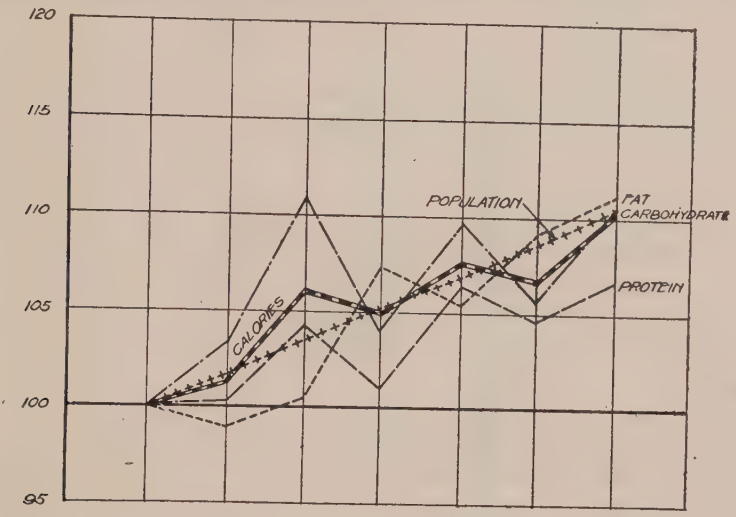

FIG. 65. RELATIVE CURVES FOR HUMAN FOOD CONSUMPTION

The figure for the year 1911–1912 is taken as 100 in each case and the relative figure for each year calculated to that base.

TABLE 115

Consumption of human foods, primary and secondary, relative to 1911–12, taken as 100

	POPULATION	PROTEIN	FAT	CARBOHYDRATE	CALORIES (MILLIONS)
1911–12	100.0	100.0	100.0	100.0	100.0
1912–13	101.7	100.2	99.0	103.4	101.4
1913–14	103.4	104.3	100.3	110.7	106.1
1914–15	105.1	101.3	107.3	104.0	104.9
1915–16	106.8	106.3	105.3	109.6	107.6
1916–17	108.5	104.5	109.1	105.5	106.7
1917–18	110.2	106.4	110.9	110.2	110.0
Average, whole period	105.1	103.3	104.6	106.2	105.2
Average, 1911–12 to 1916–17	104.3	102.8	103.5	105.5	104.5

of all propaganda from dietary cranks and from commercial interests, it is clear that the American people depend to an overwhelming degree upon animal sources for their fat intake, rather than upon vegetable oils, nuts

and the like. This condition is naturally reversed as regards carbohydrate. Ninety-five per cent of this nutrient comes from primary sources and only 5 from secondary. In the total nutritional calory intake 61 per cent comes from primary foods and 39 per cent from secondary.

It is interesting to compare the percentage of American nutritional intake derived from primary and secondary sources with corresponding British figures. Calculating roughly from table I of the official British report[8] on the subject I find that 42 per cent of the protein intake, 92 per cent of the fat intake, and 35 per cent of the energy value of the total nourishment of the population of the United Kingdom comes from *secondary* sources. In other words, the British get less of their protein and calories and more of their fat from animal products, exclusive of fish, than the Americans do. The differences, however, are not very great, indicating generally similar dietary habits in the two populations, a fact which we know on general grounds to be true.

The above comparisons regarding primary and secondary sources of human food are shown graphically in figures 66 and 67.

The next problem concerns the relative proportion of the total nutritional intake furnished by the several different large food commodity classes. The data on this point for the main groups are collected in tables 116 to 119 and 121 to 124 inclusive. The arrangement of these tables is to give first the annual average for the six years preceding the entrance of the United States into the war, and then to give 1917–1918, our first year in the war, separately. The reason for such a time division is obvious. There is no reason to suppose that the consumption of food in this country was affected by the war till the time we entered and the United States Food Administration began its work. Before then the population had gone on consuming food at about the usual normal rate. There was no reason or incentive to do otherwise, except in so far as price had an influence. But in 1917–1918 a wholly new and extraordinary influence was brought into play to alter the national food habits. This was the Food Administration, which through its recommendations, on the one hand, and regulations on the other hand, sought to modify the consumption rate of certain commodities and succeeded in doing so, as will presently appear in detail.

In tables 116 to 119 the percentage figures are first given separately and then accumulated to 100 in another column.

The data of tables 116 to 119 are shown graphically in figure 68.

[8] The food supply of the United Kingdom, a report drawn up by a Committee of the Royal Society at the request of the Board of Trade, London (Cd. 8421), 1917, pp. 35.

From these tables and diagrams it is seen that the grains stand at the head of the list in contribution of protein, carbohydrate and calories. Meats come first in contribution of fat, second in protein and calories. Thirty-six

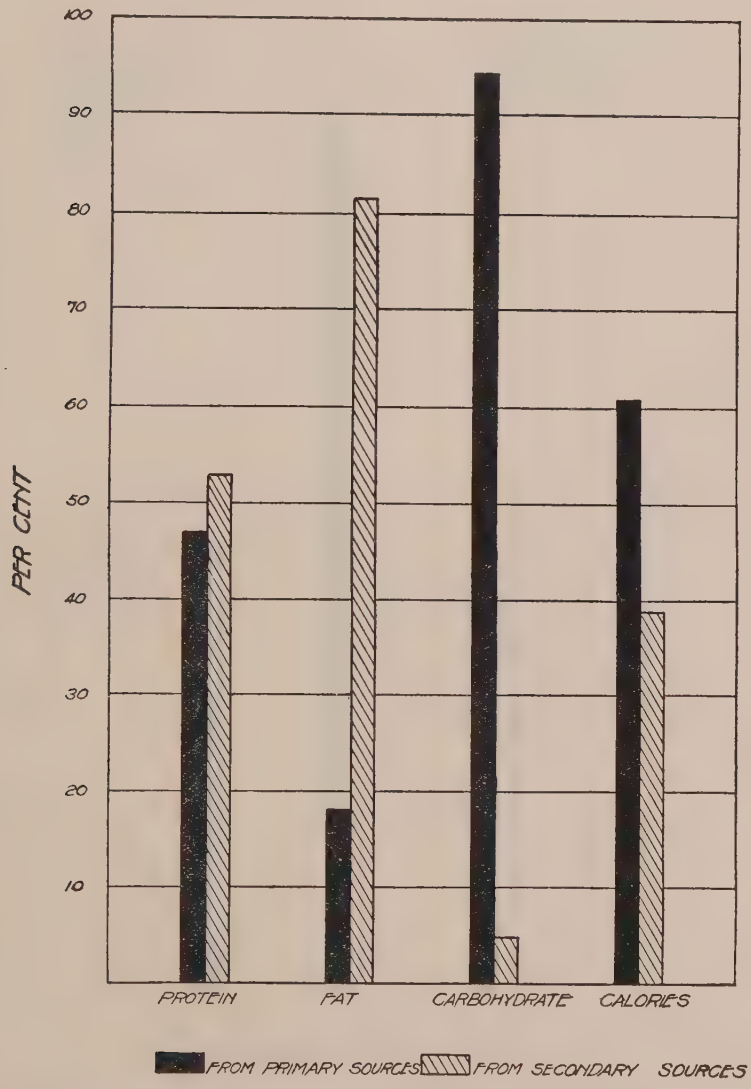

FIG. 66. DIAGRAM SHOWING THE PERCENTAGES OF THE TOTAL NUTRITIONAL INTAKE OF THE AMERICAN PEOPLE DERIVED FROM PRIMARY AND SECONDARY SOURCES

per cent of our protein intake normally is in the form of grain, 26 per cent in meats and 20 per cent in dairy products. These three great commodity groups together make up nearly 83 per cent of our total protein intake.

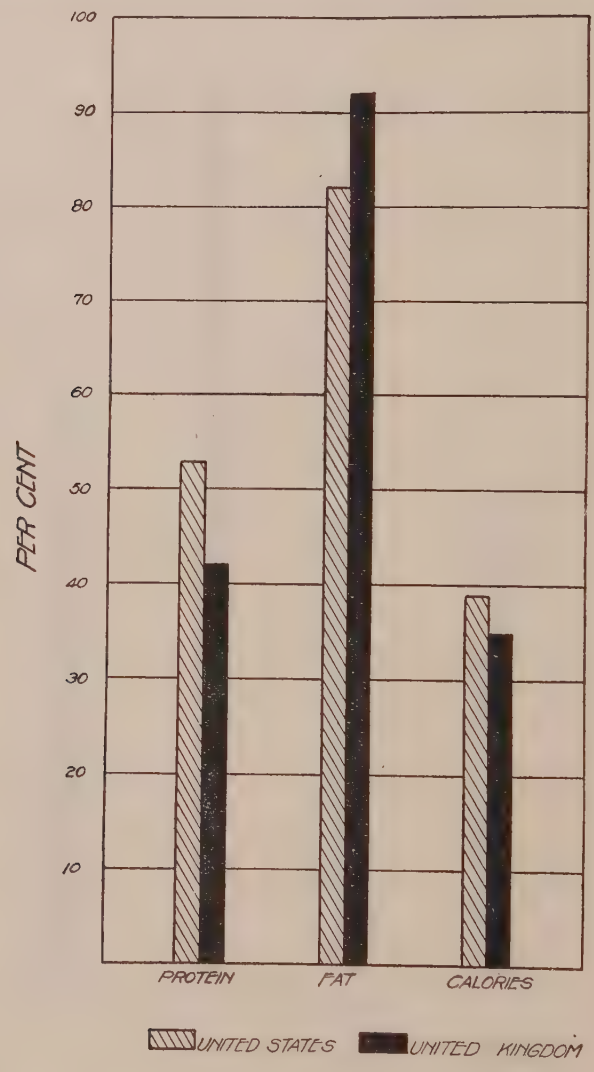

FIG. 67. DIAGRAM SHOWING THE RELATIVE PROPORTIONS OF THE AMERICAN AND BRITISH FOOD INTAKE DERIVED FROM ANIMAL SOURCES (EXCLUSIVE OF FISH)

The total consumption of human foods was higher in 1917–1918 than the average of the preceding six years. This is to be expected from increase of

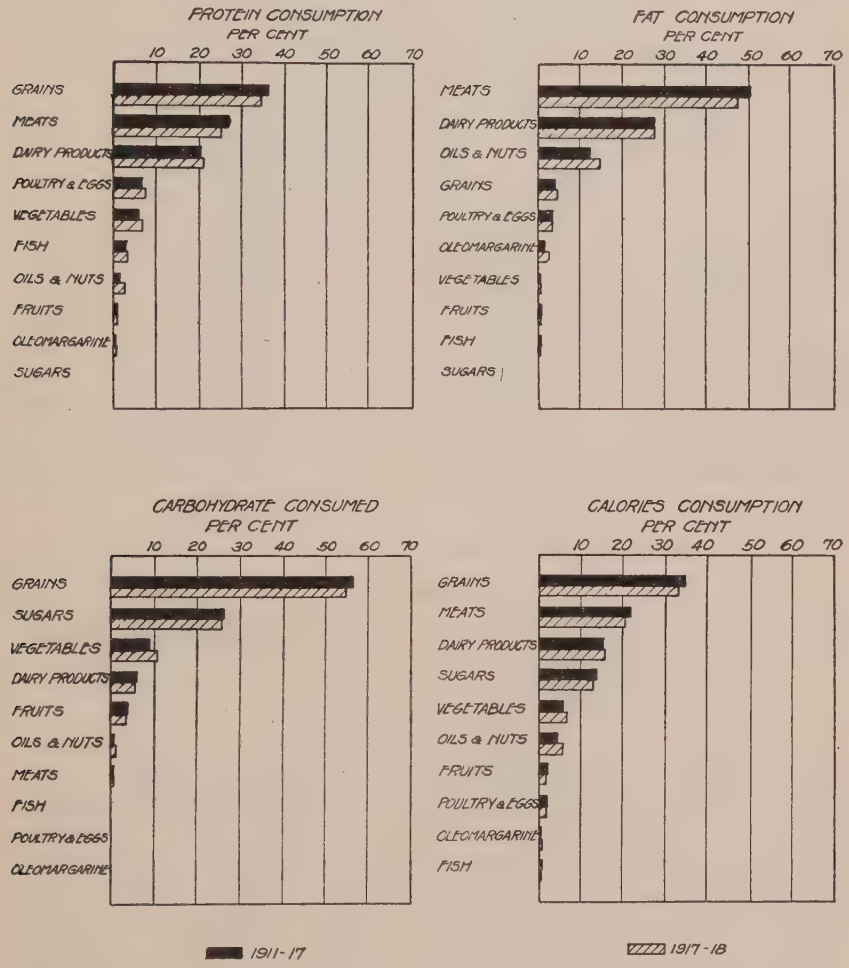

FIG. 68. SHOWING THE PERCENTAGE CONTRIBUTION OF THE DIFFERENT GREAT FOOD COMMODITY GROUPS TO THE NUTRITIONAL INTAKE OF THE UNITED STATES FOR (*a*) THE AVERAGE YEAR OF THE PERIOD 1911–1912 TO 1916–1917 AND (*b*) 1917–1918

population and from the greater prosperity of the people incident to high wages, etc. But the proportion of the total contributed by the grains and meats was smaller in 1917–1918. In other words, the two great commodity

TABLE 116

Consumption of protein in human foods, primary and secondary in the United States arranged by groups in order of magnitude

GROUP	AVERAGE FOR THE SIX YEARS, 1911–12 TO 1916–17			FOR 1917–18		
	Absolute consumption of protein (metric tons)	Percentage consumption	Cumulated per cent	Absolute consumption of protein (metric tons)	Per centage consumption	Cumulated per cent
Grains	1,316,140	36.01	36.01	1,303,348	34.44	34.44
Meats	964,117	26.38	62.39	945,277	24.98	59.42
Dairy products	744,784	20.38	82.77	788,969	20.85	80.27
Poultry and eggs	246,178	6.74	89.51	248,772	6.57	86.84
Vegetables	214,404	5.87	95.38	285,413	7.54	94.38
Fish	84,852	2.32	97.70	85,021	2.25	96 63
Oils and nuts	57,839	1.58	99.28	102,022	2.69	99.32
Fruits	24,965	0.69	99.97	23,621	0.62	99.94
Oleomargarine	838	0.02	99.99	1,808	0.05	99 99
Sugars	455	0.01	100.00	439	0.01	100.00
Total	3,654,572	100.00		3,784,690	100.00	

TABLE 117

Consumption of fat in human foods, primary and secondary, in the United States, arranged by groups in order of magnitude.

GROUP	AVERAGE FOR THE SIX YEARS, 1911–12 TO 1916–17			FOR 1917–18		
	Absolute consumption of fat (metric tons)	Percentage consumption	Cumulated per cent	Absolute consumption of fat (metric tons)	Percentage consumption	Cumulated per cent
Meats	2,591,613	50.66	50.66	2,602,187	47.48	47.48
Dairy products	1,405,918	27.49	78.15	1,505,129	27.46	74.94
Oils and nuts	623,385	12.19	90.34	781,461	14.26	89.20
Grains	203,585	3.98	94.32	227,807	4.16	93.36
Poultry and eggs	173,349	3.39	97.71	175,220	3.20	96.56
Oleomargarine	57,965	1.13	98.84	125,024	2.28	98.84
Vegetables	21,126	0.41	99.25	27,864	0.51	99.35
Fruits	20,242	0.40	99.65	17,381	0.32	99.67
Fish	18,096	0.35	100.00	17,866	0.33	100.00
Sugars	0	0	100.00	0	0	100.00
Total	5,115,279	100.00		5,479,939	100.00	

TABLE 118

Consumption of carbohydrates in human foods, primary and secondary, in the United States, arranged by groups in order of magnitude

GROUP	FOR THE SIX YEARS, 1911–12 TO 1916–17			FOR 1917–18		
	Absolute consumption of carbohydrate (metric tons)	Percentage consumption	Cumulated per cent	Absolute consumption of carbohydrate (metric tons)	Percentage consumption	Cumulated per cent
Grains	9,208,939	56.11	56.11	9,291,445	54.22	54.22
Sugars	4,193,095	25.55	81.66	4,374,194	25.53	79.75
Vegetables	1,421,851	8.66	90.32	1,844,682	10.76	90.51
Dairy products	899,691	5.48	95.80	917,169	5.35	95.86
Fruits	627,487	3.82	99.62	601,350	3.51	99.37
Oils and nuts	57,097	0.35	99.97	102,231	0.60	99.97
Meats	4,907	0.03	100.00	4,717	0.03	100.00
Fish	20	0.0001	100.00	25	0.0001	100.00
Poultry and eggs	0	0	100.00	0	0	100.00
Oleomargarine	0	0	100.00	0	0	100.00
Total	16,413,087	100.00		17,135,813	100.00	

TABLE 119

Consumption of human foods, primary and secondary, in the United States, in terms of caloric value, arranged by groups in order of magnitude

GROUP	FOR THE SIX YEARS, 1911–12 TO 1916–17			FOR 1917–18		
	Absolute consumption (million calories)	Percentage consumption	Cumulated per cent	Absolute consumption (million calories)	Percentage consumption	Cumulated per cent
Grains	45,057,265	34.68	34.68	45,569,323	33.31	33.31
Meats	28,104,069	21.63	56.31	28,122,722	20.55	53.86
Dairy products	19,834,010	15.26	71.57	21,010,397	15.36	69.22
Sugars	17,196,595	13.24	84.81	17,939,129	13.11	82.33
Vegetables	6,910,026	5.32	90.13	8,999,132	6.58	88.91
Oils and nuts	6,269,270	4.82	94.95	8,104,157	5.92	94.83
Fruits	2,862,540	2.20	97.15	2,722,604	1.99	96.82
Poultry and eggs	2,620,311	2.02	99.17	2,648,262	1.93	98.75
Oleomargarine	542,719	0.42	99.59	1,170,593	0.86	99.61
Fish	534,509	0.41	100.00	533,419	0.39	100.00
Total	129,931,314	100.00		136,819,738	100.00	

groups on which the most stress was laid in the conservation campaign of 1917–1918 show a reduction in the part which they play in national nutrition. The effect of the conservation work will, however, be more clearly shown when we come to the consideration of individual commodities.

Of the fat normally consumed 51 per cent is furnished by the meats as a group; 27 per cent by the dairy products; and 12 per cent by the vegetable oils and nuts. The grains normally furnish 3.98 per cent of the fat intake and in 1917–1918 this rose slightly to 4.16, due to the increased consumption of corn meal.

TABLE 120

Showing the changes in food consumption in the United States in 1917–18 as compared with the average annual consumption in six preceding years (millions of calories)

GROUP	INCREASE OF CONSUMPTION IN 1917–18 OVER 6 YEAR AVERAGE	DECREASE OF CONSUMPTION IN 1917–18 UNDER 6 YEAR AVERAGE	PERCENTAGE INCREASE	PERCENTAGE DECREASE
Grains	512,058		1.14	
Meats	18,653		0.07	
Dairy products	1,176,387		5.93	
Sugars	742,534		4.32	
Vegetables	2,089,106		30.23	
Oils and nuts	1,834,887		29.37	
Fruits		139,936		4.89
Poultry and eggs	27,951		1.07	
Oleomargarine	627,874		115.69	
Fish		1,090		0.20
Total	6,888,424		5.30	
Population	5,662,979		5.73	

The sugars stand second in the list as contributors of carbohydrate to consumption, with 26 per cent of the total, to which 56 per cent is furnished by the grains. Of the remainder of the carbohydrate intake vegetables normally contribute about 9 per cent, the dairy products 5 per cent, and the fruits 4 per cent.

The energy values of the groups are especially interesting as furnishing a general index of food values. Of the total energy furnished by the human food consumed 35 per cent comes from the grains, 22 per cent from the meats, 15 per cent from the dairy products and 13 per cent from the sugars. These four groups make up about 85 per cent of the total energy value of all the food consumed. Vegetables contribute only about 5 per cent, fruit and poultry about 2 per cent each, and vegetable oils and nuts nearly 5 per cent.

On the basis of table 119 it is of interest to examine somewhat more carefully the changes in consumption rate in 1917–1918 as compared with the average of the six preceding years. Such a comparison is made in table 120 and shown graphically in figure 69.

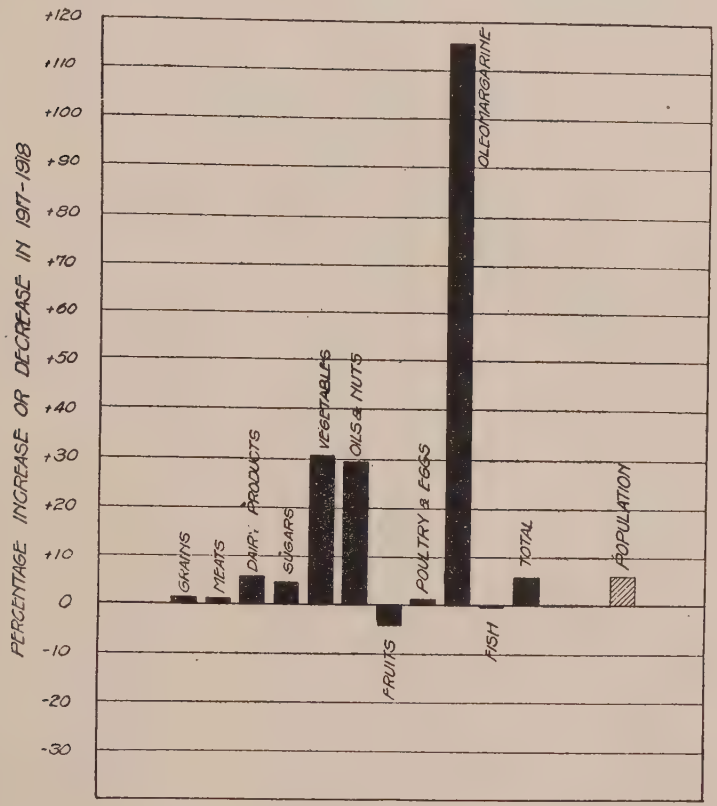

FIG. 69. DIAGRAM SHOWING THE INCREASE (OR DECREASE) IN FOOD CONSUMPTION IN 1917–1918 AS COMPARED WITH THE AVERAGE OF THE PRECEDING SIX YEARS

From table 120 and the diagram it is observed that the total increase in human food consumption in 1917–1918 was less (nearly 0.5 per cent) proportionately than the increase in population, both being compared with the average of the six preceding years. The consumption of meats practically did not increase at all, and the consumption of grains only about 1 per cent.

The great increases were first in the consumption of vegetables and oils and nuts, amounting to 30 per cent in the one case and 29 per cent in the other, and second in oleomargarine where the consumption increased nearly 116 per cent in 1917–1918 over the average of the preceding six years. In the case of vegetables and oils and nuts the increased consumption in 1917–

TABLE 121

Consumption of protein in human food, primary and secondary, in the United States, arranged by commodities in order of magnitude

ORDER NUMBER	COMMODITY	AVERAGE FOR THE SIX YEARS, 1911–12 TO 1916–17			FOR 1917–18		
		Absolute consumption of protein (metric tons)	Percentage consumption	Cumulated per cent	Absolute consumption of protein (metric tons)	Percentage consumption	Cumulated per cent
1	Wheat	1,054,548	28.85	28.85	940,543	24.85	24.85
2	Dairy products	744,784	20.38	49.23	788,969	20.85	45.70
3	Beef	528,709	14.47	63.70	539,703	14.26	59.96
4	Pork	392,665	10.74	74.44	378,799	10.01	69.97
5	Poultry and eggs	246,178	6.74	81.18	248,772	6.57	76.54
6	Corn	202,717	5.55	86.73	242,395	6.40	82.94
7	Potatoes	114,598	3.14	89.87	143,167	3.78	86.72
8	Fish	84,852	2.32	92.19	85,021	2.25	88.97
9	Legumes	69,669	1.91	94.10	105,578	2.79	91.76
10	Nuts	46,819	1.28	95.38	81,939	2.16	93.92
11	Mutton	43,712	1.20	96.58	28,298	0.75	94.67
12	Other cereals	30,471	0.83	97.41	65,088	1.72	96.39
13	Other vegetables	30,137	0.82	98.23	36,668	0.97	97.36
14	Rice	17,231	0.47	98.70	30,725	0.81	98.17
15	Rye	11,174	0.31	99.01	24,597	0.65	98.82
16	Cocoa	11,020	0.30	99.31	20,083	0.53	99.35
17	Apples	8,719	0.24	99.55	7,458	0.20	99.55
18	Other fruits	7,620	0.21	99.76	9,283	0.25	99.80
19	Bananas	6,979	0.19	99.95	5,771	0.15	99.95
20	Oranges	1,647	0.04	99.99	1,109	0.03	99.98
21	Oleomargarine	838	0.02	100.01	1,808	0.05	100.03
22	Sugars	455	0.01	100.02	439	0.01	100.04
	Oils	0	0	100.02	0	0	100.04
Total		3,654,572			3,784,690		

1918 is probably to be attributed largely to the activity of the Food Administration in urging the consumption of these commodities to afford a relief of the pressure on wheat and meat products. In the case of oleomargarine the increased consumption is clearly due entirely to a favorable price differential as compared with butter and lard, taking into account palatability.

The only two great commodity groups showing decreases in consumption in 1917–1918 are fruits and fish. In both cases the result is probably to be explained by price influences, taken together with palatability and popular ideas as to relative necessity in the diet. For example the price of meat may rise relatively much more than that of fruits or fish without leading to

TABLE 122

Consumption of fat in human foods, primary and secondary, in the United States, arranged by commodities in order of magnitude

ORDER NUMBER	COMMODITY	AVERAGE FOR THE SIX YEARS, 1911–12 TO 1916–17			FOR 1917–18		
		Absolute consumption of fat (metric tons)	Percentage consumption	Cumulated per cent	Absolute consumption of fat (metric tons)	Percentage consumption	Cumulated per cent
1	Pork	2,024,236	39.57	39.57	2,045,653	37.33	37.33
2	Dairy products	1,405,918	27.49	67.06	1,505,129	27.46	64.79
3	Oils	505,201	9.88	76.94	554,851	10.13	74.92
4	Beef	505,033	9.87	86.81	513,596	9.37	84.29
5	Poultry and eggs	173,349	3.39	90.20	175,220	3.20	87.49
6	Corn	97,355	1.90	92.10	118,845	2.17	89.66
7	Nuts	92,348	1.81	93.91	179,337	3.27	92.93
8	Wheat	91,929	1.80	95.71	81,835	1.49	94.42
9	Mutton	65,499	1.28	96.99	46,853	0.86	95.28
10	Oleomargarine	57,965	1.13	98.12	125,024	2.28	97.56
11	Cocoa	25,836	0.51	98.63	47,273	0.86	98.42
12	Fish	18,096	0.35	98.98	17,866	0.33	98.75
13	Other cereals	12,391	0.24	99.22	23,104	0.42	99.17
14	Other vegetables	9,935	0.19	99.41	12,586	0.23	99.40
15	Apples	8,629	0.17	99.58	7,451	0.14	99.54
16	Other fruits	7,713	0.15	99.73	6,767	0.12	99.66
17	Potatoes	6,366	0.12	99.85	7,953	0.15	99.81
18	Legumes	4,824	0.09	99.94	7,325	0.13	99.94
19	Bananas	3,488	0.07	100.01	2,885	0.05	99.99
20	Rye	1,479	0.03	100.04	3,256	0.06	100 05
21	Rice	431	0.01	100.05	767	0.01	100.06
22	Oranges	411	0.01	100.06	278	0.01	100.07
	Sugars	0	0	100.06	0	0	100.07
Total		5,115,279			5,479,939		

any reduction in consumption, owing to the general belief that meat is a more necessary article of diet than the other two sorts of food mentioned.

We may then consider the gross consumption of individual commodities on the same plan that has just been used in handling the groups. The data are given in tables 121 to 124 inclusive. In these tables it will be noted that

the cumulated percentage columns run to more than 100 per cent by trifling amounts. This is to take care of the item "other meat products" which appears in the net export table but not in production (in basic tables not here given). In the main consumption table it is carried into the subtotal

TABLE 123

Consumption of carbohydrate in human foods, primary and secondary, in the United States arranged by commodities in order of magnitude

ORDER NUMBER	COMMODITY	AVERAGE FOR THE SIX YEARS, 1911–12 TO 1916–17			FOR 1917–8		
		Absolute consumption of carbohydrate (metric tons)	Percentage consumption	Cumulated per cent	Absolute consumption of carbohydrate (metric tons)	Percentage consumption	Cumulated per cent
1	Wheat	6,944,794	42.31	42.31	6,195,182	36.15	36.15
2	Sugars	4,193,095	25.55	67.86	4,374,194	25.53	61.68
3	Corn	1,805,564	11.00	78.86	2,155,310	12.58	74.26
4	Potatoes	935,881	5.70	84.56	1,169,204	6.82	81.08
5	Dairy products	899,691	5.48	90.04	917,169	5.35	86.43
6	Apples	312,589	1.90	91.94	268,425	1.57	88.00
7	Other vegetables	303,868	1.85	93.79	398,275	2.32	90.32
8	Legumes	182,103	1.11	94.90	277,203	1.62	91.94
9	Other fruits	171,574	1.05	95.95	219,237	1.28	93.22
10	Rice	170,151	1.04	96.99	303,428	1.77	94.99
11	Other cereals	159,113	0.97	97.96	352,857	2.06	97.05
12	Rye	129,318	0.79	98.75	284,668	1.66	98.71
13	Bananas	111,628	0.68	99.43	92,328	0.54	99.25
14	Nuts	35,571	0.22	99.65	63,054	0.37	99.62
15	Oranges	31,696	0.19	99.84	21,360	0.12	99.74
16	Cocoa	21,526	0.13	99.97	39,177	0.23	99.97
17	Pork	2,740	0.02	99.99	2,859	0.02	99.99
18	Beef	1,707	0.01	100.00	1,577	0.01	100.00
19	Mutton	484	0.0029	100.00	315	0.0018	100.00
20	Fish	20	0.0001	100.00	25	0.0001	100.00
	Oils	0	0	100.00	0	0	100.00
	Poultry and eggs	0	0	100.00	0	0	100.00
	Oleomargarine	0	0	100.00	0	0	100.00
Total		16,413,087			17,135,813		

"Meats," but does not appear as a separate item, because of the impossibility of calculating it as such.

The data of tables 121 to 124 inclusive are exhibited graphically in figures 70 to 73.

Taking first the protein consumption, as given in table 121, we see that wheat stands at the head of the list as a source of protein for the population of this country, contributing nearly 29 per cent normally to the total. Dairy products are second with 20 per cent of the total. Beef with 14 per cent and pork with 11 per cent stand next. The other commodities con-

TABLE 124

Consumption of human foods, primary and secondary, in the United States, in terms of caloric value, arranged by commodities in order of magnitude

ORDER NUMBER	COMMODITY	AVERAGE FOR THE SIX YEARS, 1911–12 TO 1916–7			FOR 1917–18		
		Absolute consumption (million calories)	Percentage consumption	Cumulated per cent	Absolute consumption (million calories)	Percentage consumption	Cumulated per cent
1	Wheat	33,657,299	25.90	25.90	30,021,979	21.94	21.94
2	Pork	20,453,649	15.74	41.64	20,594,616	15.05	36.99
3	Dairy products	19,834,010	15.26	56.90	21,010,397	15.36	52.35
4	Sugars	17,196,595	13.24	70.14	17,939,129	13.11	65.46
5	Corn	9,141,678	7.03	77.17	10,938,521	7.99	73.45
6	Beef	6,892,851	5.30	82.47	7,017,398	5 13	78.58
7	Oils	4,700,590	3.62	86.09	5,162,528	3.77	82.35
8	Potatoes	4,366,750	3.36	89.45	5,455,418	3.99	86.34
9	Poultry and eggs	2,620,311	2.02	91.47	2,648,262	1.94	88.28
10	Other vegetables	1,465,344	1.13	92.60	1,904,998	1.39	89.67
11	Apples	1,403,750	1.08	93.68	1,205,454	0.88	90.55
12	Nuts	1,196,911	0.92	94.60	2,262,988	1.65	92.20
13	Legumes	1,077,932	0.83	95.43	1,638,716	1.20	93.40
14	Other cereals	893,383	0.69	96.12	1,927,964	1.41	94.81
15	Other fruits	800,831	0.62	96.74	994,221	0.73	95.54
16	Mutton	791,032	0.61	97.35	553,498	0.40	95.94
17	Rice	774,430	0.60	97.95	1,381,039	1.01	96.95
18	Rye	590,475	0.45	98.40	1,299,820	0.95	97.90
19	Oleomargarine	542,719	0.42	98.82	1,170,593	0.86	98.76
20	Fish	534,509	0.41	99.23	533,419	0.39	99.15
21	Bananas	519,109	0.40	99.63	429,360	0.31	99.46
22	Cocoa	371,769	0.29	99.92	678,641	0.50	99.96
23	Oranges	138,850	0.11	100.03	93,569	0.07	100.03
Total		129,931,314			136,819,738		

tributing more than 2 per cent to the total protein intake of the population are, in the order named: Poultry and eggs, corn, potatoes, and fish. Taken together, these eight commodities furnish 92 per cent of the total protein intake. We see that a very few commodities furnish a very large percentage of the nutritional intake. This fact, in and of itself, helps enormously

towards the possibility of making a study such as this substantially accurate in its results. It is clear that the minor items omitted from the calculations have no significance in the final general result. If four food commodities furnish nearly 75 per cent of the total protein ingested, it is obvious that a

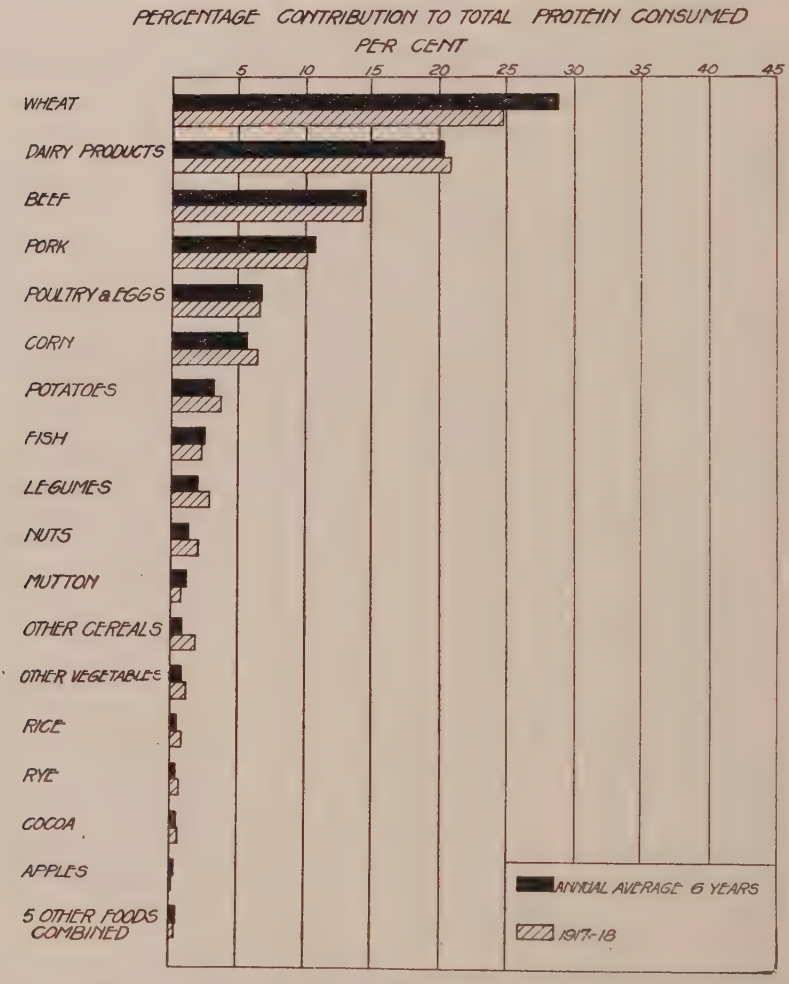

FIG. 70. DIAGRAM SHOWING THE PERCENTAGE OF THE TOTAL PROTEIN CONSUMED IN THE UNITED STATES CONTRIBUTED BY EACH OF 23 COMMODITIES

The solid bars denote the average consumption in the six years 1911–1912 to 1916–1917. The cross-hatched bars denote the consumption in 1917–1918.

large error, or even the entire omission, of single ones of the other minor items can have but little effect.

Comparing the order of the commodities in 1917–1918 with the average of the six preceding years, it is seen that the only change of position among the eight commodities normally furnishing over 90 per cent of the protein

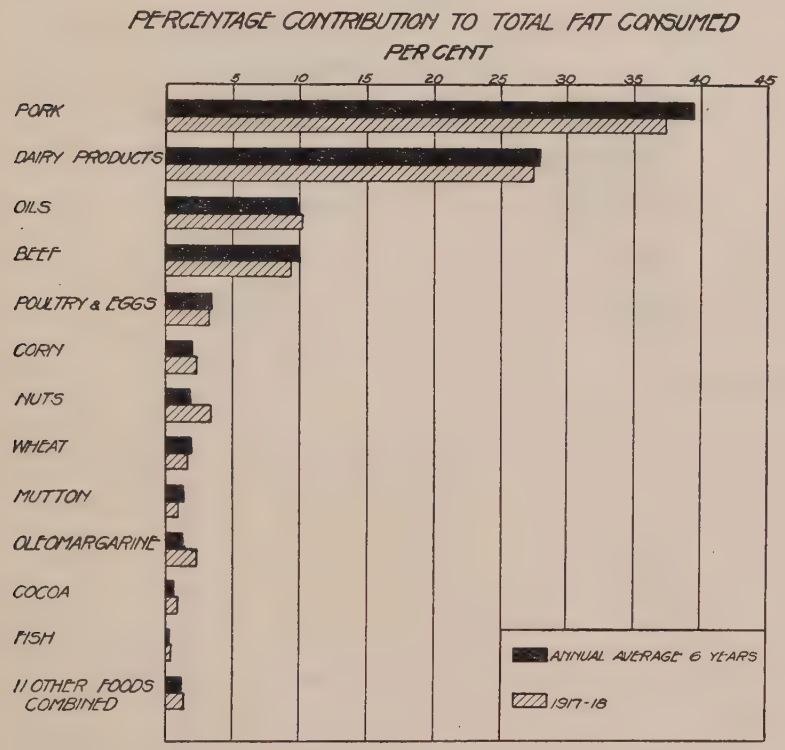

FIG. 71. DIAGRAM SHOWING THE PERCENTAGE OF THE TOTAL FAT CONSUMED IN THE UNITED STATES CONTRIBUTED BY EACH OF 23 COMMODITIES

The solid bars denote the average consumption in the six years 1911–1912 to 1916–1917. The cross-hatched bars denote consumption in 1917–1918.

is in respect of the last one on the list, namely, fish. In 1917–1918 the legumes (beans and peas) moved up to the eighth place and fish moved to the ninth place.

Turning to the fat consumption, it is seen that approximately 40 per cent of the total fat in the nutritional intake of this country comes from pork and its products. The hog is in a class by itself as a source of fat for human

nutrition, with the population of this country. Dairy products stand second in the list, with approximately 27.5 per cent of the total. After the dairy products there is a considerable drop in percentage contribution as we pass to the next item on the list, namely, the vegetable oils, which normally furnish only about 10 per cent of the fat intake. Beef contributes almost

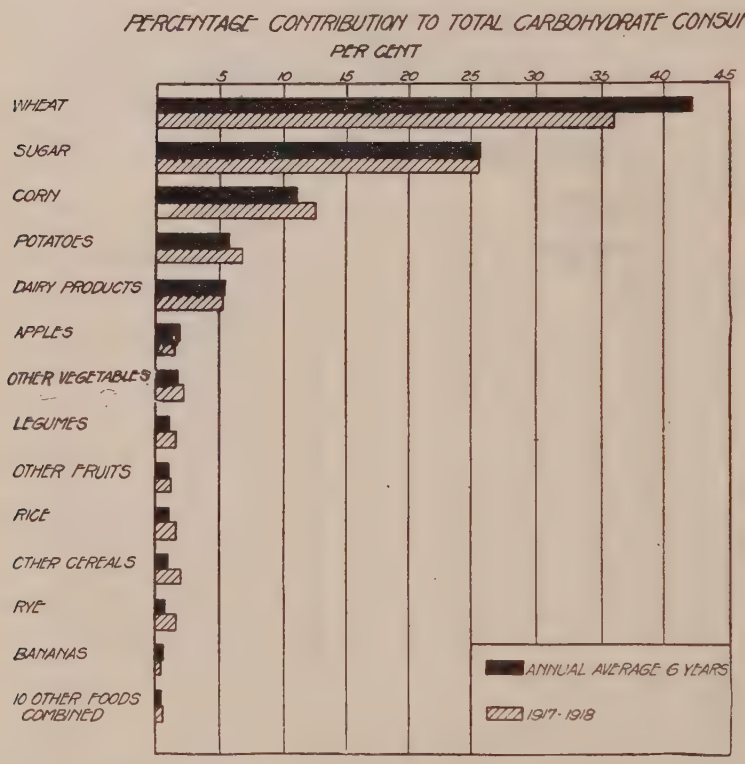

FIG. 72. DIAGRAM SHOWING THE PERCENTAGE OF THE TOTAL CARBOHYDRATE CONSUMED IN THE UNITED STATES CONTRIBUTED BY EACH OF 23 COMMODITIES

The solid bars denote the average consumption in the six years 1911–1912 to 1916–1917. The cross-hatched bars denote the consumption in 1917–1918.

exactly the same percentage. The four commodities named together furnish nearly 87 per cent of the total fat intake. Only one other commodity group—namely, poultry and eggs—furnishes more than 2 per cent normally.

In 1917—1918 there are some changes of significance in the relative position of the commodities as fat contributors. The first four items, pork,

dairy products, oils and beef, stand in the same order in 1917–1918 as in the six years preceding. Nuts moved up in 1917–1918 to the fifth place from the seventh, which they had occupied before. Oleomargarine moved from the tenth place to the seventh. Corn, in spite of the increased consumption in

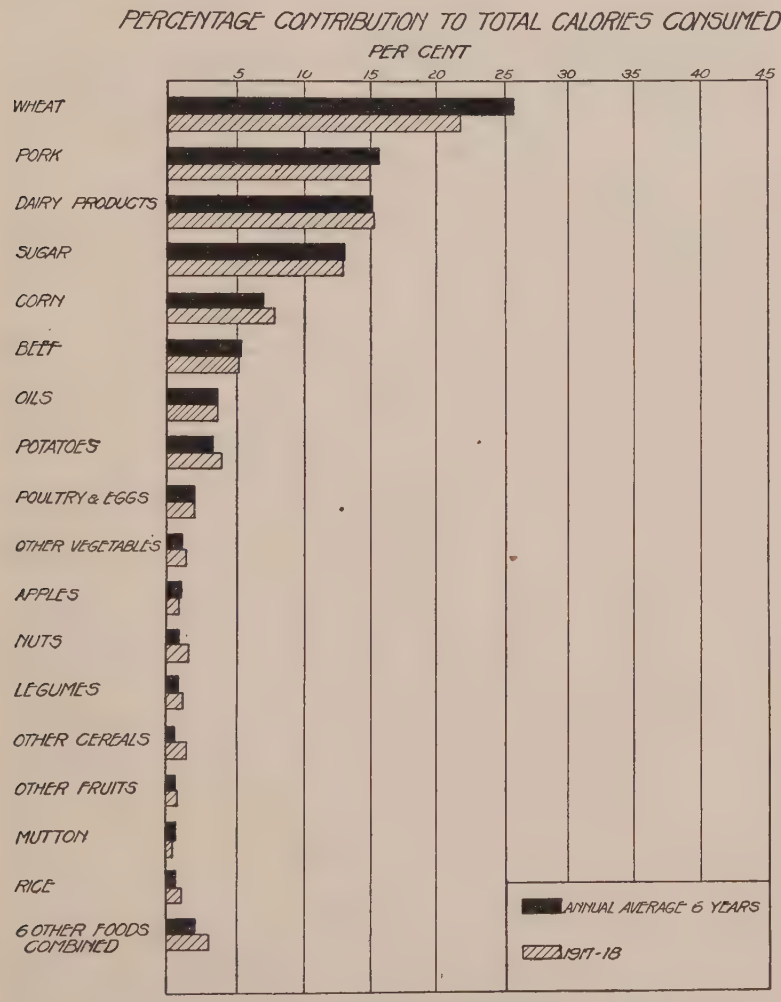

FIG. 73. DIAGRAM SHOWING THE PERCENTAGE OF THE TOTAL ENERGY VALUE OF THE FOOD CONSUMED IN THE UNITED STATES CONTRIBUTED BY EACH OF 23 COMMODITIES

The solid bars denote the average consumption in the six years 1911–1912 to 1916–1917. The cross-hatched bars denote the consumption in 1917–1918.

1917–1918, dropped from the sixth place to the eighth in percentage contribution. Twelve of the great commodity groups before our entry into the war, and thirteen in 1917–1918, contribute less than 1 per cent to the total fat intake.

In carbohydrate consumption wheat stands at the head of the list with over 42 per cent normally. The sugars stand second with about 26 per cent, and corn with 11 stands next. These three commodities, together with potatoes and the dairy products, contribute altogether 90 per cent of the carbohydrate intake. There is no change in the relative position of the commodities falling in the 90 per cent group in 1917–1918 as compared with the average of the six preceding years.

A noteworthy feature of this table 123, dealing with carbohydrates, is the relative position of the sugars. Many persons regard sugar as a pleasant but not essential part of the dietary. It is obvious enough that this is a mistaken point of view. Any commodity which furnishes nearly 26 per cent of the carbohydrate intake of the population must be regarded as an important essential. To get an idea of the importance of the sugar relatively it is only necessary to compare it with some of the items farther down in the table. For example, we see that the sugars contribute more than twenty times as much to the carbohydrate intake of the nation as does rice

In table 124 we get a summarized view of the general nutritional importance of the several food commodities, because here we are dealing with the energy content as measured in calories. The order of the products in this table may be taken as the general order of nutritional significance of the great staple foods in this country. Wheat stands at the head of the list, contributing nearly 26 per cent to the total. Pork comes next with normally 16 per cent, and dairy products third with 15 per cent, and the sugars fourth with 13 per cent. Then follow corn, beef, the vegetable oils, potatoes, poultry and eggs. These nine commodity groups together make up over 91 per cent of the total nutritional intake of the population. The smallest contribution to the total nutrition is made by oranges, furnishing about 0.1 per cent to the total. Bananas and fish furnish only about 0.4 per cent of the total, and rye and rice only a little more.

The changes in 1917–1918, as compared with the average in the six preceding years, as shown in table 124, are extremely interesting. The figures show in much more detail than any that are available the precise effects of the conservation and substitution campaign of the United States Food Administration during 1917–1918. While wheat normally contributes 25.9 per cent of the total nutritional intake (as measured by energy value), in 1917–1918 it contributed but 21.9 per cent. To go farther down the table,

rice, which normally contributed but 0.6 per cent to the total nutritional intake, contributed 1 per cent in 1917–1918.

The changes in consumption, as indicated in table 124, are of such great interest that it is worth while to examine them more in detail. To this end a table on the same plan as table 120 is shown.

TABLE 125

Showing the changes in food consumption in the United States in 1917–18 as compared with the average annual consumption of six preceding years for 23 staple human foods (millions of calories)

COMMODITY	INCREASE OF CONSUMPTION IN 1917–18 OVER 6 YEAR AVERAGE	DECREASE OF CONSUMPTION IN 1917–18 UNDER 6 YEAR AVERAGE	PERCENTAGE INCREASE	PERCENTAGE DECREASE
Wheat		3,635,320		10.80
Pork	140,967		0.69	
Dairy products	1,176,387		5.93	
Sugar	742,534		4.32	
Corn	1,796,843		19.66	
Beef	224,547		3.26	
Oils	461,938		9.83	
Potatoes	1,088,668		24.93	
Poultry and eggs	27,951		1.07	
Other vegetables	439,654		30.00	
Apples		198,296		14.13
Nuts	1,066,077		89.07	
Legumes	560,784		52.02	
Other cereals	1,034,581		115.80	
Other fruits	193,390		24.15	
Mutton		237,534		30.03
Rice	606,609		78.33	
Rye	709,345		120.13	
Oleomargarine	627,874		115.69	
Fish		1,090		0.20
Bananas		89,749		17.29
Cocoa	306,872		82.54	
Oranges		45,281		32.61
Total net increase	6,888,424		5.30	
Population	5,662,979		5.73	

The data of table 125 are exhibited graphically in figure 74. In this diagram the total length of the bars from the *O* line shows the total percentage increase or decrease in consumption in 1917–1918 as compared with the preceding six years. The cross-hatched portion of each bar shows the percentage increase in population, and therefore the part of the increased

consumption to be expected as a result of population increase. Where the black bar is below the top of the cross-hatched population bar it means a conservation. Thus the true conservation on wheat amounted to 10.80 + 5.73 = 16.53 per cent of the normal average consumption.

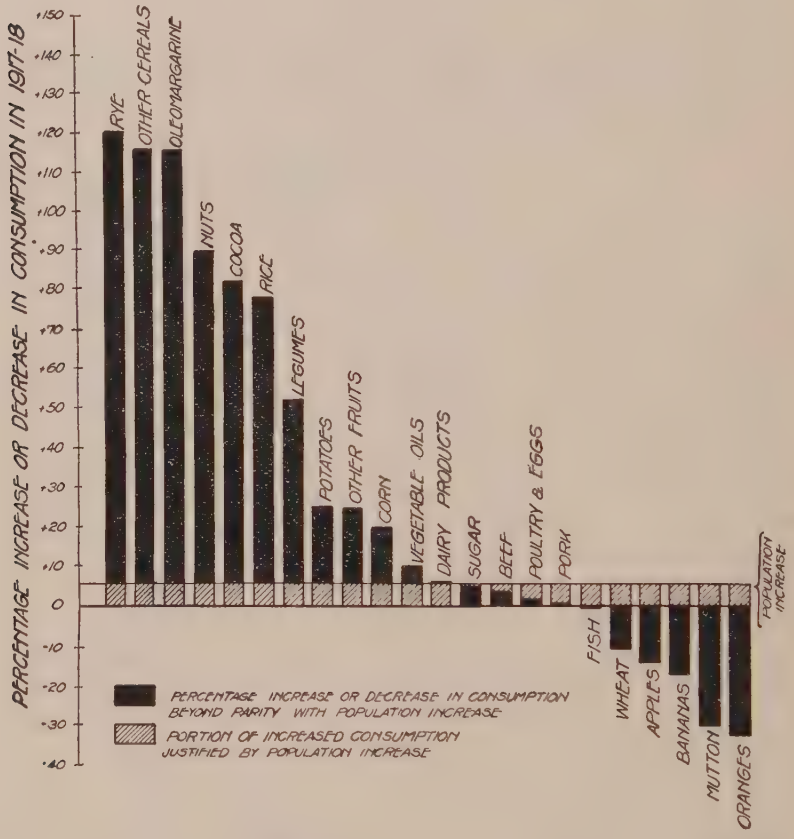

FIG. 74. SHOWING THE PERCENTAGE INCREASE (OR DECREASE) IN CONSUMPTION IN 1917–1918 AS COMPARED WITH THE ANNUAL AVERAGE OF THE SIX YEARS PRECEDING FOR EXPLANATION SEE TEXT

The table and diagram bring out very clearly the effectiveness of the Food Administration's campaign for conservation and substitution in foods. It will be noted at once that the commodities showing great increases in consumption in 1917–1918 over the preceding years are, for the most part, those which the Food Administration urged to be substituted for articles

of which the supply was less abundant, and for which the needs of the Allies were greater. Thus, rye, which constituted the most popular of the substitutes for wheat in the public mind, shows the greatest increased consumption in 1917–1918. Next to it stands the "other cereals" of our classification, including barley and buckwheat. Nuts, rice and the vegetables generally show increases beyond the population increase, showing that the people very generally followed the suggestions of the Food Administration to consume more of these products and save wheat. The articles on which the Food Administration most strongly urged conservation—namely, wheat, beef, mutton, pork and the sugars—all show either a consumption actually below the normal average, or else a very slight absolute increase well below the population percentage increase. In either case a real and substantial conservation is, of course, shown. The decrease in consumption of the most popular fruits, oranges, apples and bananas is largely if not entirely explained by high prices for these products.

We get now to the most interesting stage of any discussion of food, namely, the per capita per day consumption. Calculating the results on this basis puts them in a form where we may form a better judgment of their meaning and compare them with accepted dietary standards.

In reducing consumption data to a per capita basis it would obviously be foolish to take the actual total population as a base, for the reason that the amount of food consumed changes with the age of the individual, particularly in early life. On account of this fact the usual practice in computations of this kind is to reduce, not to a per capita basis, but to an adult man basis. In doing this a fractional factor is used to multiply the number of individuals of certain lower ages, the magnitude of the factor being proportional to the relation which the nutritional intake of the individual at the younger age bears to that of an average adult man.

In the present study the following age-intake factors were used:

AGE IN YEARS	MAN VALUE FACTOR
0–5	0 50
6–13	0.77
14–18, male	1 00
14–18, female	0.83
19 on, male	1.00
19 on, female	0.83

These "man factor" values were adopted only after careful study of the subject. They differ in detail somewhat from those adopted by English

physiologists in similar calculations, but in the net end result come to much the same thing.

Applying these factors to the total population of the United States, and assuming that the age distribution of the population is the same in each of the years studied, we get the population in terms of adult men as set forth in table 126 for the midyear point of each of the years included in this study. These population equivalents are used as the base for the per capita per diem calculations which follow.

Before entering on the detailed discussion of per capita consumption figures it is well to recall a fact which is liable to escape attention, unless special attention is called to it. This is that the final figures in this discussion, which are called "consumption figures," really include something more than consumption in a nutritional sense. They involve the food actually eaten plus that which is wasted by loss in cooking, in garbage, etc.

TABLE 126

Population of continental United States in terms of adult men

YEAR	POPULATION EQUIVALENT IN ADULT MEN, JANUARY 1
1912	79,571,000
1913	80,930,000
1914	82,289,000
1915	83,648,000
1916	85,007,000
1917	86,366,000
1918	87,724,000

It is necessary to be entirely clear on this point. In calculating the nutrients use has been made of factors which allowed for *inedible* refuse, so that all such portions of the foods (as produced or imported) have already been deducted in the calculations up to this point. Furthermore, gross losses from storage, spoilage, transportation, etc., have been deducted. Even after all these deductions have been made, however, it is obvious that there is still a considerable amount of loss and wastage of strictly edible material, which might be saved and consumed under a theoretically ideal system of preparing food for the table plus a conscientious ingestion of every bit of edible material. Of course, as a matter of fact, neither of these theoretically ideal conditions at all prevail. There is a considerable loss of nutrient values in the process of cooking as ordinarily practiced. This loss is undoubtedly greater for fats than for any other of the nutrients. It is a troublesome and time-consuming process for the housewife to conserve and utilize all of the fat which gets melted and floats about in the water in which foods

are cooked, or adheres to the utensils. Nor, in the minds of most people, is there any necessity or desirability of saving this fat. In fact, a great many people in this country object very strongly to what they designate as "greasy cooking." Consequently, floating fat of soup stock is skimmed off and thrown away in the vast majority of instances. The result is that in calculations made in the way those of this study have been made, which include the total nutrient value in the edible portion of food materials, after deducting inedible waste and the losses which accrue up to the time the food reaches the consumer, there is bound to be an apparently high consumption of fats. The figures here presented are really statements of consumption plus edible waste and should be so regarded.

Another important factor is that of edible waste in garbage: that is to say, the uneaten portion of the prepared food which is edible and might be consumed, but is not for reasons of taste, over-estimation of ingestive capacity, etc.

It is quite impossible to arrive at any accurate estimate of the amount of the losses of nutrients in cooking and in avoidable wastage of edible material. On the first point it would be extremely difficult ever to gather accurate data because the practice of housewives and cooks varies so enormously in this regard. That a great deal can be accomplished in reducing the amount of edible material going into the garbage can was demonstrated with both the civilian and the Army population of the United States during 1917 and 1918.[9]

The study of Murlin gives the data regarding edible waste obtained from the nutritional surveys of the training camps. The average figures for 213 messes show that 7 per cent of the protein supplied was wasted, 9 per cent of the fat and 6 per cent of the carbohydrate. Because of special conditions surrounding the investigation, however, and because of the differences of camp life, these figures are not at all applicable to civilian conditions.

Looking at the matter from the national point of view, it seems probable that of the protein in human foods left in the country for consumption in the statistical sense, it is safe to say that 5 per cent is lost in edible wastage; of the fat left in the country for consumption as human food, it is believed that at least 25 per cent is lost through wastage. This figure seems large, but it probably under-estimates rather than over-estimates the fact. Of the carbohydrates, probably there is 20 per cent of edible wastage.

[9] See Chap. XV, *infra*, and Murlin, J. H., Diet of the United States Army soldier in the training camp, *Jour. Amer. Med. Assoc.*, vol. 71, pp. 950–951, 1918.

The total statistical consumption (ingestion plus edible wastage) of human food in the United States, by years from 1911 to 1918, is shown on an "adult man" per capita basis in table 127.

Applying the estimated percentage deductions for edible wastage stated above to the per capita average for the whole period we have the following results for ingested human food:

114 grams protein per man per day
127 grams fat per man per day
433 grams carbohydrate per man per day
3424 calories per man per day

TABLE 127

Summary of consumption per adult man

YEAR	PROTEIN		FAT		CARBOHYDRATE		CALORIES	
	Per annum	Per day	Per annum	Per day	Per annum	Per day	Per annum	Per day
	kilos	*grams*	*kilos*	*grams*	*kilos*	*grams*		
1911–12	44.70	122	62.12	170	195.48	536	1,563,450	4,283
1912–13	44.04	121	60.44	166	198.68	544	1,558,232	4,269
1913–14	45.08	124	60.22	165	209.25	573	1,591,621	4,361
1914–15	43.05	118	63.42	174	193.42	530	1,560,326	4,275
1915–16	44.48	122	61.22	168	200.48	549	1,574,621	4,314
1916–17	43.01	118	62.45	171	189.94	520	1,536,833	4,211
1917–18	43.14	118	62.47	171	195.34	535	1,559,661	4,273
Average, whole period	43.91	120	61.78	169	197.45	541	1,565,075	4,288
Average, 1911–12 to 1916–17	44.05	121	61.65	169	197.82	542	1,566,032	4,290

These figures are probably very close to the fact as regards protein and carbohydrate. They are undoubtedly somewhat too high still as regards fat, because the edible wastage of this component is higher than the 25 per cent used. The intention, however, has been to use the most conservative figures in estimating waste.

For purposes of comparison table 128 is inserted. This table is based upon certain American dietary studies analyzed in the writer's statistical laboratory.

The general agreement of these results with those set forth in the present study, which were reached by totally different procedure, is evident. The statistical estimate of per capita protein consumption over the whole population is distinctly higher than in this small group. The fat consumption is higher but not by so large an amount as protein. The farmers and profes-

sional men show a higher net energy intake than the general average for the whole country, which would, of course, be expected. Mechanics are about the same as the average for the country in energy intake.

In any case there is one fact which must not be lost sight of, namely, that while the figures of table 127 do in fact represent ingestion *and* waste, it still is true, and the constancy of the figures in successive years proves its truth, that to maintain naturally a contented feeling in respect of nutrition the population *actually uses up* the amounts of nutrients indicated in table 127. To make these gross consumption figures materially less would require a profound readjustment of the dietary and culinary habits of the people,

TABLE 128

Summary of some dietary studies in 11 groups and 116 families

	NUMBER OF FAMILIES	AVERAGE YEARLY INCOME	DAYS PER MAN	PER MAN PER DAY			
				Protein	Fat	Carbohydrate	Energy
				grams	*grams*	*grams*	*calories*
Mother wage earners	8	$640	212	105	65	472	2,895
Garment makers	7	724	168	109	81	495	3,145
Laborers	6	1,497	305	94	102	479	3,210
Retired	5	1,647	130	81	121	420	3,095
Clerks (office)	11	1,934	225	92	120	419	3,125
Mechanics	8	2,133	259	97	113	460	3,245
Teachers	32	2,150	620	88	125	430	3,195
Professional men	17	2,208	438	99	148	438	3,480
Engineers (professional)	5	2,253	97	85	128	395	3,070
Salesmen	5	2,527	121	90	111	405	2,980
Farmers	12		384	102	131	506	3,610
Average	116	$1,771*	260	95	113	447	3,185

* Average of 104 families (farmers excluded).

fixed by centuries of usage. Discussion of the minimum protein, fat and carbohydrate requirements of a nation are in considerable degree academic if they base themselves upon net consumption rather than gross consumption. A considerable excess over any agreed upon minimum *physiological* requirements must always be allowed, because there will inevitably be, in fact, a margin between actual gross consumption and net physiological ingestion or utilization. The present study, through the figures summarized in table 127 gives a clearer and probably more nearly exact picture of what this margin between net and gross consumption must be, in a population of the habits of the American people, than has hitherto been available. It

may well be theoretically true that a man needs only 75 grams or 50 grams of protein per day to sustain life and health, but in actual fact the American man *uses up*, in one way or another, about 120 grams a day. Furthermore, if the seven years' experience is any criterion, he will continue to use up

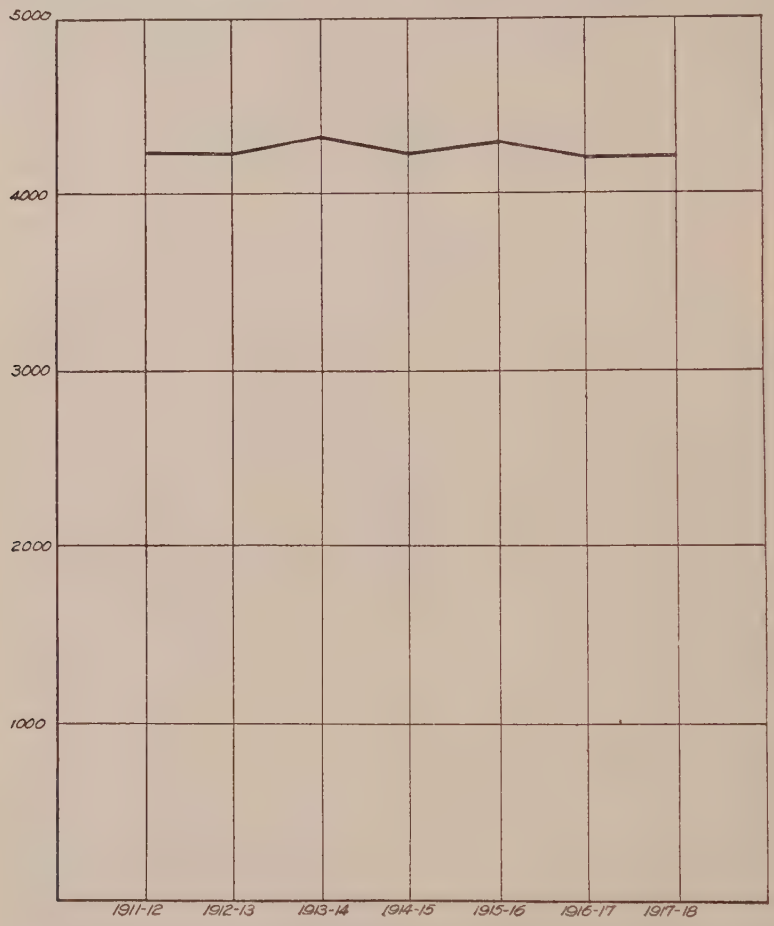

FIG. 75. DIAGRAM SHOWING THE ENERGY VALUE IN CALORIES OF THE GROSS CONSUMPTION OF HUMAN FOOD PER ADULT MAN PER DAY

about 120 grams per diem until such time as his general habits of life are in some manner rather profoundly changed. Doubtless they can be changed. But until they are, one must count on supplying about 120 grams of protein per day to each man equivalent component of the population.

The data of table 127 for calories are shown graphically in figure 75.

From this diagram it is apparent that there has been only a very slight decrease in per capita gross food consumption since 1911. Even this probably does not mean that the population is eating any less, but that because of the gradually rising prices through all this period there has been a narrowing of the margin between gross and net consumption; or, put in another way, there has been a slight reduction in the wastage of edible foods.

CHAPTER XV

Food Waste[1]

In July, 1917, the writer inaugurated in the Statistical Division of the United States Food Administration a system of voluntary statistical returns from the leading cities in the country, regarding the amount of garbage collected monthly and, where possible, the amount of grease recovered from the garbage collected. The purpose underlying the plan was to obtain information which would serve the officials of the Food Administration as an index of the effectiveness of their propaganda campaign urging the people to avoid waste in the preparation and use of food. In view of the somewhat novel character of the statistical material which has been collected in this work, it seemed desirable to give it permanent record. Accordingly there are presented here the records of two complete years, from May, 1916, to April, 1918, inclusive.

The statistical material was obtained through the voluntary coöperation of municipal officials. In the first instance the mayors of all the larger cities in the country were asked if they would not arrange to have the proper official in their municipalities make a monthly report to the Food Administration on the amount, in tons, of garbage collected each month in the current year and the corresponding month of the previous year, beginning with May, 1917. The response was very gratifying, particularly in regard to the willingness, not to say eagerness, to coöperate, of those asked. In a rather considerable number of cases it developed, apparently quite as much to the astonishment of the city officials as to ours, that the city had no record, nor any ready method of finding out how much garbage was collected in that city in a given interval of time. Finally, however, we were able to get 96 cities, with an estimated aggregate population of over 26,000,000 reporting regularly and for each month in the two years from May, 1916, to May, 1918. These cities include roughly about one-fourth of all the people living in this country. The numbers are sufficiently large to give considerable trustworthiness to the data as indicative of urban conditions in the country in general.

[1] This chapter is based upon a paper entitled "Statistics of garbage collection and garbage grease recovery in American cities," published in *Jour. Indust. and Engineer. Chem.*, vol. 10, no. 11, p. 927, 1918.

At the end of the year a tabulation of all the monthly returns which had been made was returned to the reporting city official for verification or correction. In this way it is believed that the figures here given are accurate so far as concerns the reporting of the municipal records. The original records themselves in some cases obviously do not include the whole of the garbage produced. In a few they are grotesquely far from the mark. It is, for example, inconceivable that the hundred odd thousand people who live in Nashville, Tenn., *produce* only about 400 tons of garbage in a year, while about an equal number, say 10,000 fewer, of people living in Norfolk, Va., produce in the neighborhood of 30,000 tons in a year. The fact is that the figures given refer to tonnage of garbage officially *collected* either by or under the official control of the municipality so that the amount is a matter of city record. Only in cities where by ordinance it is forbidden to dispose of garbage in any other way than by delivery to the organized official collecting agency of the city can the statistics here given be regarded as representing the total amount produced.

The fact that the figures are for collection rather than production does not invalidate relative comparisons of one year with another, provided of course that the scope of official collection did not change in the period. Pains have been taken to make sure by correspondence that no such changes in the plan of collection came in in the cities dealt with during the period covered.

The basic statistics are contained in table 129, in which the 96 cities covered are listed alphabetically. The data given are (*a*) population, (*b*) gross tonnage of garbage collected in 1917–1918, (*c*) gross tonnage of garbage collected in 1916–1917, fiscal year ending April 30 being taken in both cases, (*d*) a relative figure which expresses the 1917–1918 collection as a percentage of 1916–1917 collection for the same city.

The totals of this table show that in the 96 cities included in the tabulation 10 per cent less garbage was collected in 1917–1918 than in the previous year. The figures demonstrate a genuine conservation of food by the urban population of the country during that year, in the sense that 10 per cent of the usual wastage in the preparation of food and in the incomplete usage of food after its preparation was eliminated. The gross tonnage figures do not, however, give a true picture of the real amount of conservation or of the effectiveness of the Food Administration's teachings. This can only be demonstrated by the grease figures to which we shall come presently.

Of the 96 cities included in table 129, 81 showed smaller collections in 1917–1918 than in 1916–1917, and 15 had larger collections. The distribution of relative figures for these 81 cities was that shown in table 130.

TABLE 129

Total garbage collections, by tons, from 96 cities for the 2 years May 1917–April 1918 and May 1916–April 1917

CITY	POPULATION	TONS COLLECTED		RELATIVE FIGURE 1917–18 TO 1916–17 TAKEN AS 100
		May 1917 to April 1918	May 1916 to April 1917	
Akron, Ohio	150,000*	10,084	8,529	118.2
Allentown, Pa	80,000*	12,591	12,340	102.0
Atlanta, Ga	196,000*	42,412	44,798	94.7
Atlantic City, N. J	57,660	15,373	18,792	81.8
Augusta, Ga	41,040	47,480	49,338	96.2
Aurora, Ill	33,022	1,509	1,805	83.6
Baltimore, Md	593,000*	34,685	37,915	91.5
Berkeley, Cal	66,000*	8,874	9,726	91.2
Boston, Mass	781,628*	46,335	52,650	88.0
Bridgeport, Conn	172,113*	18,166	19,897	91.3
Brockton, Mass	67,449	4,117	5,794	71.1
Buffalo, N. Y	468,558	15,382	21,817	70.5
Cambridge, Mass	112,981	7,138	7,605	93.9
Cedar Rapids, Iowa	40,667§	2,282	2,230	102.3
Charleston, S. C	61,041†	26,900	32,114	83.8
Charlotte, N. C	50,000*	9,410	8,612	109.4
Chelsea, Mass	47,000*	1,746	2,455	71.1
Chicago, Ill	2,497,722	93,235	124,496	74.9
Cincinnati, Ohio	416,300*	34,103	40,692	83.8
Cleveland, Ohio	674,073	55,466	59,708	92.9
Colorado Springs, Col	36,000*	3,832	3,974	96.4
Columbus, Ohio	220,000*	17,295	20,393	84.8
Dallas, Texas	124,537	220	267	82.4
Dayton, Ohio	155,000*	15,677	16,621	94.3
Detroit, Mich	750,000*	64,270	72,785	88.3
East Orange, N. J	39,852	3,746	3,980	94.1
El Paso, Texas	85,222*	15,948	15,969	99.9
Erie, Pa	100,000*	6,927	8,257	83.9
Everett, Mass	40,000*	1,750	1,975	88.6
Fort Wayne, Ind	81,057*	7,862	10,203	77.1
Galveston, Texas	35,000†	6,954	11,858	58.6
Grand Rapids, Mich	140,000*	7,339	8,678	84.6
Hartford, Conn	145,000*	13,829	14,644	94.4
Haverhill, Mass	51,870*	3,541	3,852	91.9
Holyoke, Mass	62,301†	1,708	2,310	73.9
Houston, Texas	165,192*	30,203	28,567	105.7
Indianapolis, Ind	271,758	19,929	23,267	85.7
Jacksonville, Fla	100,000*	17,654	24,732	71.4
Jersey City, N. J	316,889†	106,846	106,856	100.0

TABLE 129—*Continued*

CITY	POPULATION	TONS COLLECTED		RELATIVE FIGURE 1917–18 TO 1916–17 TAKEN AS 100
		May 1917 to April 1918	May 1916 to April 1917	
Joliet, Ill.	40,000*	3,232	3,636	88.9
Kansas City, Mo.	297,847	1,730	1,860	93.0
Lexington, Ky.	41,097‡	1,985	2,607	76.1
Los Angeles, Cal.	600,000	47,345	51,062	92.7
Lowell, Mass.	107,978*	3,935	4,272	92.1
Lynn, Mass.	102,425	8,591	10,153	84.6
Manchester, N. H.	85,000*	4,477	7,868	57.0
Memphis, Tenn.	148,995	15,231	17,146	88.8
Milwaukee, Wis.	474,000*	30,008	35,928	83.5
Minneapolis, Minn.	390,000*	21,041	23,307	90.3
Mobile, Ala.	60,060	19,145	19,655	97.4
Nashville, Tenn.	117,057	410	406	100.9
New Bedford, Mass.	118,158	8,774	10,162	86.3
New Orleans, La.	385,000*	83,459	98,710	84.5
Newport, Ky.	32,000	1,906	2,726	69.9
New York City, N. Y.	5,377,456*	445,237	487,451	91.3
Niagara Falls, N. Y.	60,000*	2,900	2,865	101.2
Norfolk, Va.	106,159†	27,251	31,512	86.5
Oakland, Cal.	198,604	32,610	33,787	96.5
Oklahoma City, Okla.	92,943	2,658	2,854	93.1
Pasadena, Cal.	46,500*	2,727	1,865	146.2
Passaic, N. J.	70,000*	28,987	27,599	105.0
Paterson, N. J.	138,443	31,359	31,985	98.0
Philadelphia, Pa.	1,709,518	114,160	101,678	112.3
Pittsburgh, Pa.	579,090	72,612	73,758	98.4
Pittsfield, Mass.	39,607*	1,418	1,789	79.3
Portland, Maine	63,867	5,012	7,046	71.1
Portland, Oregon	311,000*	30,674	33,876	90.5
Quincy, Mass.	45,500*	506	627	80.7
Racine, Wis.	48,000*	3,751	4,268	87.9
Reading, Pa.	112,561*	38,546	11,584	73.8
Richmond, Va.	160,000	26,188	30,942	84.6
Roanoke, Va.	40,574	4,568	6,058	75.4
Rochester, N. Y.	275,000*	25,926	30,782	84.2
Sacramento, Cal.	77,500*	16,901	23,894	70.7
St. Louis, Mo.	905,650*	8,657	44,555	86.8
St. Paul, Minn.	276,000*	14,593	17,105	85.3
Salem, Mass.	46,994	1,994	2,198	90.7
San Diego, Cal.	92,000*	173	257	67.3
San Francisco, Cal.	550,000*	131,653	150,415	87.5
San José, Cal.	40,000	4,549	4,976	91.4
Savannah, Ga.	68,805	9,262	11,516	80.4

TABLE 129—*Concluded*

CITY	POPULATION	TONS COLLECTED		RELATIVE FIGURE 1917–18 TO 1916–17 TAKEN AS 100
		May 1917 to April 1918	May 1916 to April 1917	
Schenectady, N. Y.	105,000	4,111	4,419	93.0
Scranton, Pa.	146,811	12,698	15,518	81.8
Somerville, Mass.	90,500*	5,870	5,875	99.9
Springfield, Ill.	70,000*	47,910	28,315	169.2
Springfield, Mass.	105,942	10,394	11,640	89.3
Syracuse, N. Y.	155,624	12,895	14,055	91.7
Tampa, Fla.	53,886	18,081	18,023	100.3
Terre Haute, Ind.	66,083	11,730	19,631	59.8
Toledo, Ohio	220,000*	22,180	23,971	92.5
Trenton, N. J.	111,593‡	14,751	16,166	91.2
Washington, D. C.	400,000*	46,732	46,293	100.9
Wheeling, W. Va.	50,671	4,603	5,658	81.4
Wilmington, Del.	94,265	18,986	14,187	133.8
Worcester, Mass.	187,000*	6,992	6,828	102.4
Youngstown, Ohio	120,000*	8,424	9,827	85.7
Total	26,034,685	2,388,932	2,609,134	91.6

* Population in 1918.
† Population in 1917.
‡ Population in 1916.
§ Population in 1915.

TABLE 130

Distribution of relative figures of cities showing smaller collection in 1917–18 than in 1916–17

RELATIVE FIGURE	NUMBER OF CITIES
50–59	2
60–69	2
70–79	14
80–89	31
90–99	32

Roughly speaking, three-fourths of these 81 cities had relative figures of 80 or above, indicating reduction of collections from 1 to 20 per cent. The four cities giving relative figures under 70, namely, Manchester, N. H., Galveston, Texas, Terre Haute, Ind., and San Diego, Calif., make very creditable showings indeed.

The 15 cities showing an increase in garbage collections in 1917–1918 are separately treated in table 131, which has the same arrangement as table 129.

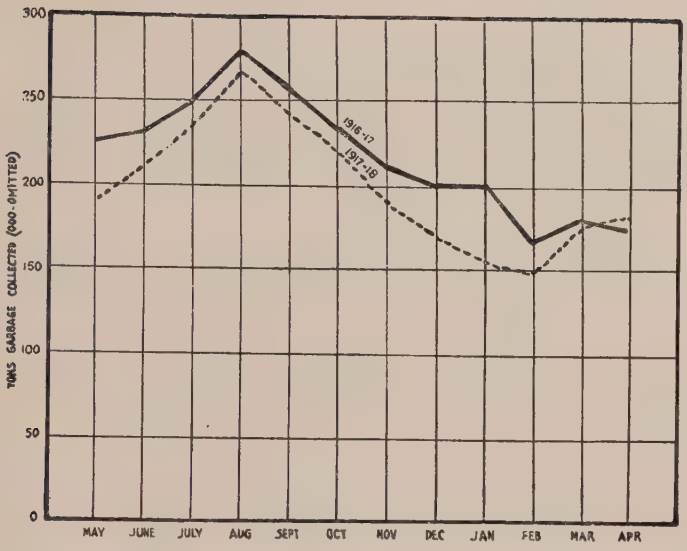

FIG. 76. THE SEASONAL CURVE OF GARBAGE PRODUCTION, BASED ON AVERAGE RETURNS FROM 96 LARGEST CITIES

TABLE 131

Fifteen cities in which the annual garbage collections increased in 1918 relative to 1917, in order of increase

ORDER	CITY	TONS COLLECTED		RELATIVE FIGURE 1917–18 TO 1916–17 TAKEN AS 100
		May 1917 to April 1918	May 1916 to April 1917	
1	Tampa, Fla.	18,081	18,023	100.3
2	Nashville, Tenn.	410	406	101
3	Washington, D. C.	46,732	46,293	101
4	Niagara Falls, N. Y.	2,900	2,865	101
5	Allentown, Pa.	12,591	12,340	102
6	Cedar Rapids, Iowa	2,282	2,230	102
7	Worcester, Mass.	6,992	6,828	102
8	Passaic, N. J.	28,987	27,599	105
9	Houston, Texas	30,203	28,567	106
10	Charlotte, N. C.	9,420	8,612	109
11	Philadelphia, Pa.	114,160	101,678	112
12	Akron, Ohio	10,084	8,529	118
13	Wilmington, Del.	18,986	14,187	134
14	Pasadena, Cal.	2,727	1,865	146
15	Springfield, Ill.	47,910	28,315	169

Of these 15 cities, the first seven may at once be dropped out of account as the increase is very small, 2 per cent or less. Of the remainder, 5, namely Passaic, Philadelphia, Akron, Wilmington and Springfield, Ill., are places which received considerable increments of population within 1917–1918 on account of war activities of one sort or another, such as munition making

TABLE 132

The 4 Industrial Cities Showing the Greatest Increase in Garbage Collection in 1918 Relative to 1917, Giving Collections by Months

	SPRINGFIELD, ILL.		WILMINGTON, DEL.		AKRON, OHIO		PHILADELPHIA, PA.	
	Tons collected	Relative	Tons collected	Relative	Tons collected	Relative	Tons collected	Relative
May, 1917.....	3,690	293	923	77	1,005	172	8,017	102
May, 1916.....	1,260		1,203		583		7,823	
June, 1917.....	3,528	280	970	83	894	148	9,588	119
June, 1916.....	1,260		1,165		604		8,053	
July, 1917.....	2,322	158	1,200	93	864	105	11,042	131
July, 1916.....	1,474		1,287		825		8,434	
August, 1917.....	2,393	224	4,120	222	1,094	113	14,883	140
August, 1916.....	1,068		1,853		967		10,604	
September, 1917....	919	124	3,582	208	1,165	118	13,690	142
September, 1916....	742		1,722		985		9,665	
October, 1917.....	2,745	235	1,436	97	1,069	129	11,183	132
October, 1916.....	1,170		1,476		828		8,498	
November, 1917....	2,650	142	1,995	183	792	96	8,174	107
November, 1916....	1,872		1,089		829		7,655	
December, 1917....	3,438	102	1,282	139	630	122	6,647	90
December, 1916....	3,375		925		515		7,423	
January, 1918.....	2,217	58	927	106	539	102	7,928	103
January, 1917.....	3,792		876		528		7,702	
February, 1918.....	3,220	66	743	89	576	133	7,054	67
February, 1917.....	4,912		838		432		10,559	
March, 1918.....	11,280	253	799	84	698	114	7,962	101
March, 1917.....	4,450		952		613		7,879	
April, 1918.....	9,508	323	1,009	126	759	93	7,992	108
April, 1917.....	2,940		801		820		7,374	

and the like. The effect of such sudden increase in population on garbage collection is obvious. To show its effect in detail, table 132 has been prepared, which gives the monthly collections for the four industrial cities showing the greatest increases in garbage collections.

Table 133 gives the monthly collections in the 10 largest cities covered in the statistics, with the relative figures for each month, comparing that month in 1917–1918 with the corresponding month in 1916–1917.

TABLE 133

Garbage collections from the 10 largest cities, giving comparisons by months

	NEW YORK, N. Y.		CHICAGO, ILL.		PHILADELPHIA, PA.		ST. LOUIS, MO.		BOSTON, MASS.		CLEVELAND, OHIO		LOS ANGELES, CAL.		BALTIMORE, MD.		PITTSBURGH, PA.		SAN FRANCISCO, CAL.	
	Tons collected	Relative	Tons collected	Relative	Tons collected	Relative	Tons collected	Relative	Tons collected	Relative	Tons collected	Relative	Tons collected	Relative	Tons collected	Relative	Tons collected	Relative	Tons collected	Relative
May, 1917	36,602	81	2,990	27	8,017	102	2,794	70	4,416	89	4,165	74	3,451	91	2,886	96	5,490	88	10,803	87
May, 1916	45,197		11,177		7,823		3,997		4,980		5,660		3,812		3,006		6,244		12,438	
June, 1917	44,590	93	8,386	65	9,588	119	3,882	92	4,042	88	4,613	89	3,454	84	3,199	97	6,019	95	10,211	87
June, 1916	48,099		12,826		8,053		4,205		4,572		5,176		4,120		3,307		6,303		11,760	
July, 1917	49,295	94	11,239	79	11,042	131	4,631	84	3,870	87	5,431	90	4,453	83	4,854	97	6,728	99	10,318	85
July, 1916	52,173		14,302		8,434		5,540		4,440		6,068		5,337		5,012		6,810		12,078	
August, 1917	51,545	97	12,583	78	14,883	140	6,247	88	4,265	91	5,680	89	5,431	95	5,063	93	7,340	96	11,846	86
August, 1916	53,368		16,093		10,604		7,078		4,680		6,384		5,729		5,473		7,652		13,780	
September, 1917	45,903	94	12,142	82	13,690	142	5,591	99	4,310	92	5,639	93	5,167	101	4,567	97	7,623	102	11,121	82
September, 1916	48,934		14,774		9,665		5,645		4,668		6,069		5,111		4,709		7,481		13,541	
October, 1917	42,971	96	11,250	90	11,183	132	3,815	94	4,033	87	5,955	109	4,440	97	3,833	97	7,440	106	11,861	88
October, 1916	44,629		12,462		8,498		4,075		4,632		5,473		4,596		4,041		7,045		13,482	
November, 1917	35,551	90	8,967	93	8,174	107	2,495	81	3,631	83	4,580	92	3,395	90	2,099	95	5,877	97	10,926	90
November, 1916	39,299		9,663		7,655		3,081		4,368		4,973		3,779		2,216		6,034		12,173	
December, 1917	28,739	83	6,661	91	6,647	90	1,896	74	3,415	79	4,165	104	3,481	90	1,787	89	4,613	79	11,414	86
December, 1916	34,691		7,280		7,423		2,575		4,332		4,012		3,862		2,011		5,840		13,347	
January, 1918	24,935	76	2,388	30	7,928	103	1,362	57	2,910	63	3,751	84	3,605	90	1,780	76	4,095	69	11,537	84
January, 1917	32,975		7,897		7,702		2,403		4,608		4,485		4,026		2,357		5,907		13,696	
February, 1918	22,350	85	4,347	74	7,054	67	1,498	90	3,093	81	3,352	100	3,246	93	985	52	5,362	117	10,211	90
February, 1917	26,399		5,904		10,559		1,668		3,812		3,366		3,498		1,899		4,586		11,313	
March, 1918	29,283	98	6,051	102	7,962	101	2,011	100	4,175	103	3,552	87	3,721	99	1,769	93	5,988	132	10,800	89
March, 1917	29,995		5,936		7,879		2,020		4,051		4,087		3,740		1,905		4,539		12,092	
April, 1918	33,650	106	6,222	101	7,992	108	2,434	107	4,175	119	4,583	116	3,501	101	1,763	89	6,037	114	10,605	99
April, 1917	31,692		6,182		7,374		2,268		3,507		3,955		3,452		1,979		5,317		10,715	

The first noteworthy feature of table 133 is the considerable variation among the different cities as to the constancy of the relative figure for the different months of the year. In some of the cities it maintains a fairly even level throughout the year, notably in Baltimore and San Francisco, and to a lesser degree, Boston. The seasonal fluctuations in savings in these cities, as indicated by the relative figures, follow rather closely the *general* seasonal distribution of the garbage collections in the cities named. Others of the cities show widely varying figures in this respect, notably in Philadelphia, where, in the course of the year, the relative figure changes all the way from 67 to 142.

TABLE 134

Total tons of garbage collected in 96 cities, by months, May 1916 to April 1918

	GARBAGE COLLECTED (TONS)		
	1917–18	1916–17	RELATIVE
May	191,129.06	226,066.56	85
June	209,937.90	230,724.72	91
July	233,853.45	245,198.66	95
August	265,409.63	278,948.91	95
September	241,317.59	258,751.64	93
October	220,943.29	234,148.73	94
November	190,012.89	209,090.07	91
December	170,391.67	200,067.75	85
January	156,711.35	200,096.45	78
February	148,785.15	167,391.84	89
March	177,392.25	181,306.00	98
April	183,119.69	177,342.50	103
Totals	2,388,931.92	2,609,133.83	92

Some general features of the seasonal distribution of garbage collection are indicated in table 133. The normal seasonal curve of garbage production, however, is better shown by the sums by months of all the cities covered in table 129. This is done in table 134, where there are exhibited the total collections of garbage for the 96 cities reporting, in each month of the two fiscal years for which reports are available.

From figure 76 and table 134 it is possible to get considerable information as to the normal distribution of the garbage production in the different months of the year. The month of maximum collection is August and the month of minimum collection is February. Following February, the curve begins to rise and goes up rather steadily along something approaching a straight line to the maximum point. The fall from the maximum point in May to the minimum point in February is again nearly a straight line.

Table 134 also enables one to see in what month the conservation propaganda was the most effective. In the months of May and June and December and January, the degree or extent of the lowering of the 1917–1918 collections, as compared with the 1916–1917 collections, is largest. During the other months of the year the curves run very closely parallel. During the last month of the fiscal year the two curves cross; that is to say, the April,

TABLE 135

Tons of garbage grease recovered in 12 cities for the 2 years May 1917–April 1918 and May 1916–April 1917

CITY	POPULATION	TONS OF GARBAGE		TONS OF GREASE RECOVERED			PERCENTAGE OF GREASE		
		May 1917–April 1918	May 1916–April 1917	May 1917–April 1918	May 1916–April 1917	Relative figure	May 1917–April 1918	May 1916–April 1917	Relative figure†
Boston, Mass.	781,628*	46,335	52,650	1,401	2,140	65	3.02	4.06	74
Buffalo, N. Y.	468,558	15,382	21,817	314	494	63	2.03	2.26	90
Chicago, Ill.	2,497,722	93,235	124,496	1,656	2,869	58	1.77	2.30	77
Cleveland, Ohio	674,073	55,466	59,708	1,415	1,821	78	2.55	3.05	84
Columbus, Ohio	220,000*	17,295	20,393	354	639	55	2.04	3.13	65
Dayton, Ohio	155,000*	15,677	16,621	250	355	70	1.59	2.13	75
Indianapolis, Ind.	271,758	19,929	23,267	454	793	57	2.27	3.40	67
New Bedford, Mass.	118,158	8,774	10,162	199	270	74	2.26	2.65	85
Pittsburgh, Pa.	579,090	72,612	73,758	1,554	2,117	73	2.14	2.87	75
Philadelphia, Pa.	1,709,518	114,160	101,678	1,178	1,161	101	1.03	1.14	90
Schenectady, N. Y.	105,000	4,111	4,419	84	91	93	2.04	2.04	100
Wilmington, Del.	94,265	18,986	14,187	49	92	53	0.25	0.65	38
Totals	7,674,770	481,962	523,156	8,908	12,842	70	1.85	2.45	76

* Population 1918.

† Relative figure expressing the monthly collection for the present year as a percentage of that of the same month last year; that is, relative figures under 100 mean smaller collections and figures over 100 mean larger collections.

1918, collections were slightly larger than the April, 1917, collections. This is probably due chiefly to the fact of an increased use in April, 1918, of various vegetable foods with a comparatively large amount of inedible refuse, which increases in turn resulted from the shortage of wheat and wheat flour. People were urged to substitute and undoubtedly did so to a very considerable extent, vegetables for the scant cereals. This was particularly

true of potato consumption. Another factor in the case is undoubtedly the increase of the population in a considerable number of the cities of the United States as a result of the war conditions, munitions making, shipbuilding, etc.

We may turn now to a consideration of the grease recovery from garbage. The grease is the profitable constituent of garbage as it is ordinarily handled. The raw material also, of course, contains valuable protein and carbohydrate, but in the usual methods of reduction the tankage from which the grease has been extracted goes to fertilizer. Unfortunately, only comparatively few cities had municipal reduction plants and were able to furnish statistics of grease recovery. Such data as it was possible to collect are exhibited in table 135. The arrangement is the same as that of the earlier tables.

The data of table 135 show in the clearest manner the remarkable effect of the conservation campaign. The 12 cities show a reduction of 30 per cent in the gross tonnage of grease recovered from garbage in 1917–1918 as compared with 1916–1917. The average percentage of grease in the garbage dropped from 2.45 to 1.85. The figures demonstrate that not only was there a quantitative conservation of food effected during 1917–1918, but also, and even more important, there was a proportionally much greater qualitative conservation. There must have been in these 12 cities a great reduction in the amount of meats and fats going into the garbage can.

The two cities showing the greatest qualitative food conservation, as indicated in garbage statistics, were Columbus, Ohio, and Wilmington, Del., with relative figures of 55 and 53, respectively. In these two cities the garbage in 1917–1918 contained only a little more than half as much fatty material in 1917—1918 as in 1916–1917. This was truly a remarkable record.

Putting all the data together, it appears that, in so far as the sampling of cities may be considered representative of the urban portion of the country as a whole, there was a substantial conservation of food by the American people during 1917–1918. A reduction of 10 per cent in the gross tonnage of garbage, and of 30 per cent in the tonnage of fat recovered can only have been accomplished by a real and widespread saving and utilization of food materials which ordinarily go into the garbage can.

CHAPTER XVI

An Index of the Age Distribution of a Population[1]

It is an obvious fact that the crude death rate of any community is influenced in a marked degree by the age distribution of the living population. Before any critical conclusions about the true force of mortality can be drawn some sort of correction must be applied to take account of the age distribution of the population. If one desires to make any analysis of the correlation of death rates with each other or with sundry environmental factors it is an absolutely essential prerequisite that there be found some single numerical expression which will be an approximate index of the age distribution of the population of each locality dealt with.

From a mathematical viewpoint the problem presented is one incapable of *exact* solution. The problem is to find a single parameter which will fully describe so complex a curve as that of the age distribution of a population, the general form of which is indicated in figure 77. But this is impossible. No single parameter can possibly describe fully such a curve. The best that can be done is to approximate as closely as may be to the impossible ideal. It is the purpose of this chapter to describe and illustrate an approximation which comes very close to the requirements, indeed quite sufficiently so for all practical statistical purposes in all cases where it has yet been tested out, and is easy to calculate.

There was first proposed as an index of differences in age composition of populations, and applied to 40 American cities, the expression

$$\chi^2 = S\left(\frac{\Delta^2}{P}\right) \tag{1}$$

where Δ is the deviation for each of six age groups (viz., 0–4, 5–14, 15–24, 25–44, 45–64, 65 and over) of the percentage of the actual population of each city in 1910 in each age group, from the percentage in the same group in the Standard Population of Glover's[2] Life Table, denoted in the formula by P. S denotes summation of all six values. The value χ^2 measures the extent to which each city deviates in the age constitution of its population from a fixed standard, but does not tell the nature or kind of the deviation.

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[1] This chapter is based upon two papers, of which the first was: Pearl, R., On a single numerical index of the age distribution of a population, *Proc. Nat. Acad. Sci.*, vol. 6, pp. 427–431, 1920; and the second was: Pearl, R., and LeBlanc, T. J., A further note on the age index of a population, *ibid.*, vol. 8, pp. 300–303, 1922.

[2] Glover, J. W., *United States Life Tables*, 1910, Bureau of the Census, 1916.

The outstanding defect of this first index will, to that degree of accuracy which is requisite for all practical statistical purposes, be entirely removed by adopting as an age-constitution index the function

$$\phi = S\left\{\frac{\Delta^2}{P}\right\}(M - M_p) \qquad (2)$$

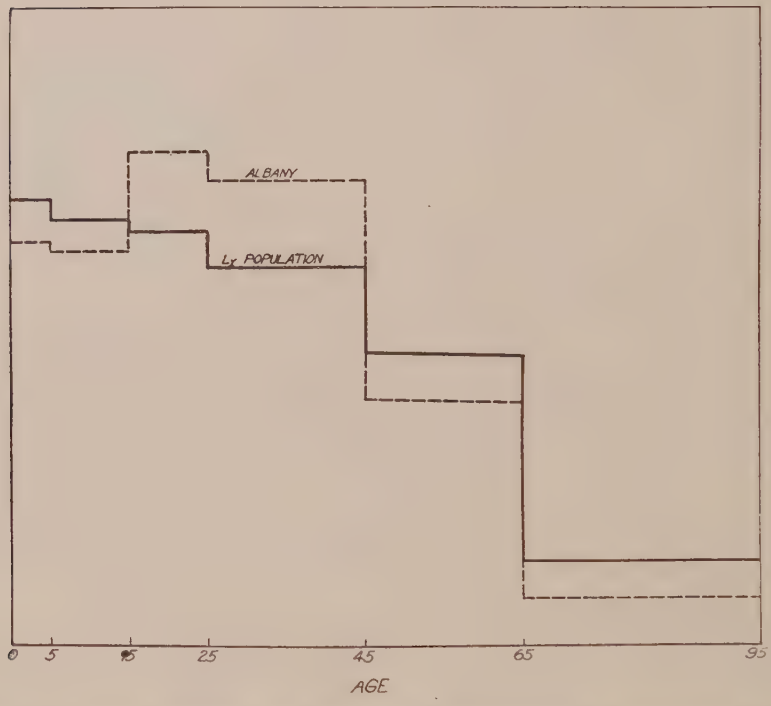

FIG. 77. DIAGRAM SHOWING AGE DISTRIBUTION OF POPULATION, GROUPED IN SIX AGE CLASSES

The last class in this and other diagrams is assumed to have its ending at age 95. The solid line gives the stationary life table population, and the broken line the population of Albany. Total area under each curve = 100 per cent.

where S, Δ and P have the same significance as before, and M = mean age of living population in any community, M_p = mean age of persons in a stationary population unaffected by migration and which, assuming the mortality rates of Glover's Life Table, would result if 100,000 persons were born alive uniformly throughout each year (M_p calculated from L_x line of Glover's Table (page 16) = 33.796 years).

This procedure simply multiplies the former index χ^2 by the difference (given its proper sign) between the mean age of the observed population and the mean age of the standard population on the basis of which χ^2 was calculated. Since in fact the mean age of any actual urban population is never likely to be as great as the mean age of the stationary population chosen as a standard of reference the actual values of ϕ will practically

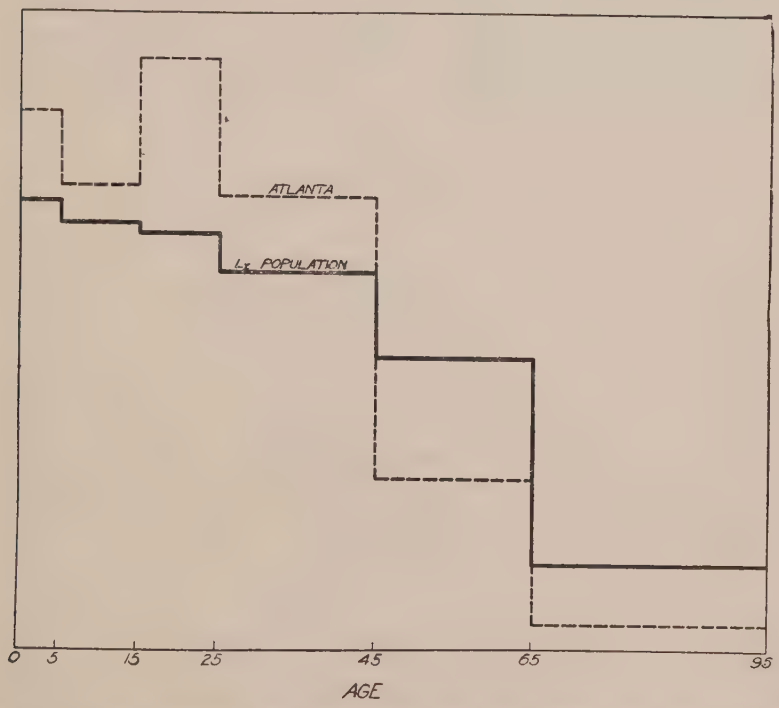

FIG. 78. DIAGRAM SHOWING AGE DISTRIBUTION OF POPULATION, GROUPED IN SIX AGE CLASSES

The solid line gives the stationary life-table population, and the broken line the population of Atlanta. Total area under each curve = 100 per cent.

always be negative for cities. The smaller these negative values are numerically the greater will be the proportion of older persons in the population concerned. In short this function ϕ tells us not only the degree to which a given population deviates in its age distribution from a fixed standard age distribution, but also the nature of this deviation, whether on the one hand in the direction of a relative excess of aged, or on the other hand in the direction of a relative excess of the young. Theoretically it is possible for

two populations differing from one another in a compensatory way to give the same values for the index ϕ. But two populations which differ in age distribution in any fundamental respect which could affect appreciably crude death rates will, in all populations so far tested, give different values of ϕ, provided the age classification from which the function is calculated is finely enough divided.

The numerical values of the index ϕ for a series of American cities, on the basis of the age distribution of the 1910 census are given by way of illustration in table 136. The cities are arranged in descending order of departure from the L_x population.

TABLE 136

Showing age-constitution indices of the population of American cities

CITY	ϕ	CITY	ϕ
Albany	−10.73	Lowell	−34.91
Los Angeles	−13.61	Philadelphia	−39.51
Oakland	−17.18	San Francisco	−43.35
Washington	−20.67	St. Louis	−44.57
Cincinnati	−22.93	Nashville	−48.32
Dayton	−24.10	Buffalo	−48.61
Indianapolis	−24.23	New Orleans	−52.44
Rochester	−26.62	Minneapolis	−55.39
Grand Rapids	−27.95	Milwaukee	−62.17
Columbus	−29.08	Newark	−62.54
Providence	−30.05	Chicago	−68.76
Cambridge	−30.26	St. Paul	−68.95
Boston	−31.05	Pittsburgh	−71.77
Baltimore	−31.80	Fall River	−73.39
Louisville	−32.19	New York	−74.42
Toledo	−33.21	Cleveland	−74.51
New Haven	−33.70	Atlanta	−82.71

The extent of the departures from the standard population denoted by the several values of the indices may be indicated graphically by plotting the age distribution curve, on a percentage basis, for the two extreme cities in the table, Albany and Atlanta, against the L_x population. This is done in figures 77 and 78.

The general characteristics of an urban population as compared with a stationary life table population are well shown in these diagrams. In general a city has a population under 40 to 45 in excess, and population older than that in defect. The reason is obvious.

It will be well to see in detail how the age distributions of some cities having nearly the same value for ϕ compare in respect of actual age dis-

tribution of their populations. Table 137 furnishes such comparisons for four pairs of cities, covering fairly the whole range of values of ϕ shown in table 136.

From this table it is seen that as we pass from cities having a value of ϕ of about 24 to cities having ϕ equal to about 74 there is a steady change from populations having relatively many persons in the higher age groups to populations having relatively few persons in those groups. Furthermore, it is evident that in each of the four pairs of cities compared the agreement between the two cities having nearly identical values of ϕ is very close in respect of actual percentage distribution of the population. Dayton and Indianapolis were for all practical statistical purposes identical

TABLE 137

Percentage age distribution of the population of certain cities in 1910

AGE CLASSES (YEARS)	DAYTON $\phi = 24.10$	INDIANAPOLIS $\phi = 24.23$	PROVIDENCE $\phi = 30.05$	CAMBRIDGE $\phi = 30.26$	NASHVILLE $\phi = 48.32$	BUFFALO $\phi = 48.61$	NEW YORK $\phi = 74.42$	CLEVELAND $\phi = 74.51$
	per cent	*per cent*	*per cent*	*per cent*	*per cent*	*per cent*	*per cent*	*per cent*
0– 4	9.1	8.0	9.7	10.3	9.2	10.0	10.6	11.1
5–14	15.4	15.3	16.5	17.5	17.8	18.3	18.1	17.4
15–24	19.5	19.4	19.0	18.4	21.9	20.6	20.8	20.5
25–44	34.6	35.9	34.2	33.3	32.2	32.3	33.9	34.4
45–64	17.0	17.0	16.2	16.0	15.1	15.5	13.7	13.4
65 and over	4.4	4.3	4.2	4.4	3.8	3.4	2.8	3.0
Totals.....	100.0	99.9	99.8	99.9	100.0	100.1	99.9	99.8

in the age distribution of their populations in 1910. So were Providence and Cambridge, Nashville and Buffalo, and New York and Cleveland. In each case the curve for one distribution of the pair winds in and out about the path set by the other. Of course, we should get finer differentiations brought out by the ϕ function if we used fifteen or twenty age classes instead of the 6 here employed. A word of caution must, however, be emphasized here. The reliability of ϕ as an approximate index of differences in age distributions of population is greater as we pass in either direction towards the ends of its range of values. In the case of populations giving values of ϕ near the mean (say in the thirties for American cities) it may be necessary, in order to get really differentiant values, to calculate ϕ from a rather fine age grouping.

Since its original proposal the index has been used in many studies, and the larger experience has strengthened our confidence in its reliability as an index of significant variations in the age constitutions of populations. But it has always been used with at least six to eight age groups covering the life span. Suppose the original statistics furnish only three age classes for the entire life span. Will this age index ϕ then give a reliable picture of the significant variations in age distribution, as we pass from city to city, or county to county?

To test this point, the obvious thing to do is to determine the correlation between the age indices for n communities, on the basis of say 3 divisions of the life span, with the age indices for the same communities on the basis of say 6 divisions of the life span. If the correlation is high it will mean that even with the coarse grouping the index is reflecting the main differences in the form of the age distributions.

A test of this sort was made. The 1915 census of Iowa[3] gives in table I the age distribution of the inhabitants of each of the 99 counties of the state in such form as to permit the formation of the following classes: 0–4, 5–9, 10–17, 18–20, 21–44, 45 and over, both figures being inclusive in each case. For each of the 99 counties ϕ was computed on the basis of this grouping of the ages. Call these values ϕ_1. Then for the same 99 counties the population was combined by addition into the 3 age groups 0–9, 10–20 and 21 and over. A new set of ϕ's was then calculated on the basis of this coarse grouping. Call these values ϕ_2. Then what we wish to know is the value of the correlation $r_{\phi_1\phi_2}$.

This was calculated by the direct product moment method, without grouping and gave as a result

$$r_{\phi_1\phi_2} = +0.84 \pm 0.02$$

It is at once evident that we are dealing here with a high degree of correlation. That the regression is linear is indicated by the spot diagram of the correlation surface shown in figure 79.

This result indicates that whenever it is impossible, by reason of unsatisfactory tabulation of the original raw statistics, to get any but a coarse age grouping, we may still use the age index ϕ with confidence that it will reflect closely the differences in population in respect of age distribution. The test here given is obviously a severe one, because all adult ages are thrown into one group. But even so, the correlation is high. With such a value of r as this, it is obvious that one could, by means of the appropriate

[3] *Census of Iowa, 1915*, published by the Executive Council.

regression equation, determine corrections for the ϕ_2 values, giving predicted or calculated ϕ_1's which would with extreme accuracy approximate the true values.

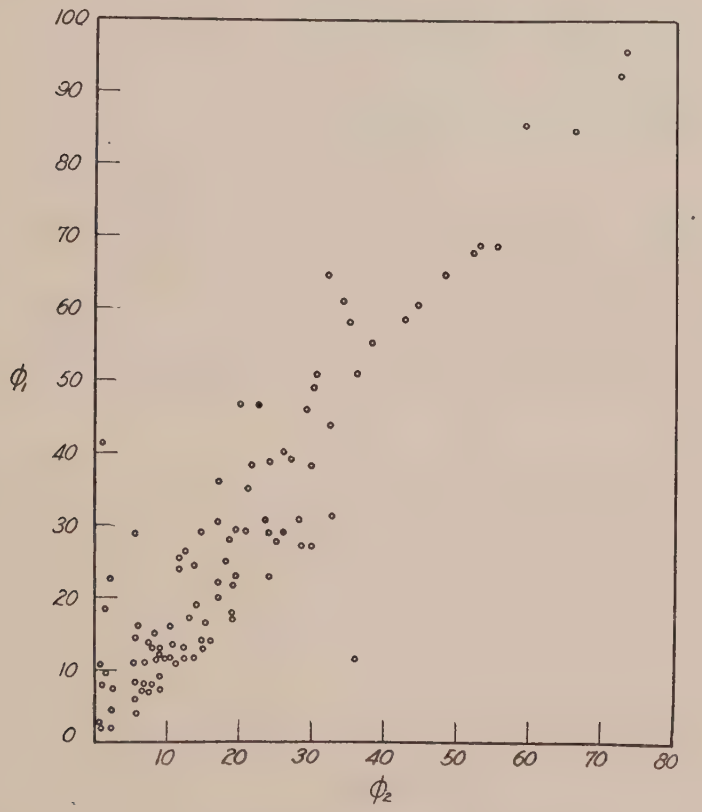

FIG. 79. SPOT DIAGRAM SHOWING THE CORRELATION BETWEEN ϕ_1 AND ϕ_2

Each dot represents one county

The means and standard deviations are as follows:

Mean ϕ_1	$= 27.27 \pm 1.46$
Standard deviation ϕ_1	$= 21.53 \pm 1.03$
Mean ϕ_2	$= 19.71 \pm 1.23$
Standard deviation ϕ_2	$= 18.14 \pm 0.87$

As would be expected the ϕ_2 mean is lower than the ϕ_1 mean, and ϕ_2 is less variable, as indicated by the standard deviation, than ϕ_1. The differ-

ence in the means is large, but this is of no great importance in the practical use of the age index. It is of much greater practical significance that the difference in the variabilities is not large. If we could trust here the theory of simple sampling (which we probably cannot) the difference between the standard deviations could not be regarded as certainly significant at all. The more important consideration is that ϕ_2 is reflecting in its standard deviation 84.2 per cent of the variation among these 99 Iowa counties in respect of age distribution of their populations, as measured by ϕ_1.

In conclusion it may be stated that since there is a high correlation between the values of ϕ for extremely coarse age groupings and the values obtained from finer groupings, this age index may be used with considerable confidence in cases where data on age distribution are available only for a few broad classes covering the life span. All our experience with this age index ϕ since its first publication has confirmed our belief that it is a function of real value to the vital statistician who wishes to make analytical studies of medical and hygienic data by modern statistical methods.

CHAPTER XVII

Some Biological Factors in the Epidemiology of Influenza[1]

The pandemic of influenza which swept over the world in 1918 was the most severe outbreak of this disease which has ever been known, and it takes an unpleasantly high rank in the roster of epidemics generally. It perhaps always will be impossible to make any precise statement of the number of people who lost their lives because of this epidemic. But it is certain that the total was an appalling one. Undoubtedly a great many more people died from this cause than from all causes directly connected with the military operations of the Great War. In the United States alone conservative estimates place the deaths from the influenza epidemic at not less than 550,000, which is approximately five times the number (111,179) of American soldiers officially stated[2] to have lost their lives from all causes in the war.

In England and Wales the curve of mortality from influenza was even in 1907, seventeen years after the epidemic of 1890, higher than it was in any of the forty years preceding 1890. The decline in the mortality rate after the 1848 epidemic in Great Britain was similarly slow.[3] There is no evident reason to suppose that conditions following the first explosion of the 1918 epidemic will be essentially different from those which obtained in the earlier cases.

For two reasons the hygienist and epidemiologist should be interested in the intensive study, from every possible angle, of the 1918 pandemic. In the first place, owing to the advances which have been made in every branch of medical science since the epidemic of 1890, there is now available a much more adequate investigational armament with which to attack the

[1] This chapter is a condensation of material contained in the following papers:

Influenza studies. I. On certain general statistical aspects of the 1918 epidemic in American cities, *Pub. Health Repts.*, vol. 34, pp. 1743–1783, 1919.

Influenza studies. II. Further data on the correlation of explosiveness of outbreak of the 1918 epidemic,

Influenza studies. III. On the correlation of destructiveness of the 1918 epidemic,

Influenza studies. IV. On the correlation between explosiveness and total destructiveness of the epidemic mortality, *ibid.*, vol. 36, pp. 273–298, 1921.

[2] As of date April 30, 1919.

[3] *Cf.* Article on Influenza in *Encyclopedia Britannica*, 11th edition, for a conveniently accessible verification of these statements.

problems raised by such an epidemic than was the case earlier. Furthermore, the whole machinery for getting accurate records of the incidence and results of the outbreak are much better now than they were thirty years ago. This is particularly true in the United States. The records of mortality connected with the 1918 epidemic are unquestionably more complete and accurate than any that have ever before been available in this country for any epidemic of anything like so great extent or force.

In the second place, the very magnitude of this epidemic was in itself a challenge to the whole medical profession. The hygienists of the world are the standing army, which is, in theory at least, maintained by society to organize and hold the defenses against such dread invaders as these. Such a blow as the last one may well inspire a slogan like that which saved Verdun, "*Ils ne passeront pas.*" If every epidemiologist does not take advantage of the present opportunity to investigate with all possible thoroughness epidemic influenza, to the end of making a better defense next time, he will have been derelict in his plain duty.

The present study was intended as a first contribution toward the statistical analysis of certain phases of the 1918 influenza epidemic. Attention will be confined entirely to the *mortality* records of some forty of the larger cities of the United States. The reason for this limitation to mortality only and to large cities is that accurate and reliable data within these limitations are now available, and the same can not be said of morbidity records, on anything like so general a scale.

The data of this study were taken primarily from the Weekly Health Index.[4] On account of varying medical opinions as to the properly reportable terminal cause of death of persons dying after having had influenza during this epidemic, it has been thought safest to use death rates from all causes for study, rather than those specifically reported to the registrar as due to influenza or pneumonia. Consequently, we shall deal with death rates from all causes in discussing the 1918 epidemic. This makes no practical difference in the statistical results, because the deviation of the curves of total mortality from their normal course during the epidemic was due entirely to causes inherently associated with the epidemic itself. The use of the death rate from all causes during the epidemic has the further advantage that it takes into account those deaths which occurred from diseases of the heart or kidneys some weeks or months after an attack of influenza from which the patient had apparently recovered, but which in reality was responsible for the fatal break-down of a part of the organic

[4] A typewritten publication issued weekly by the Bureau of the Census, and compiled under the direction of Dr. W. H. Davis, Chief for Vital Statistics.

machinery which had long been weak, and only required for its complete collapse some such strain as the attack of influenza superimposed.

The general problem with which the first part of this study will have to do is that of the statistical analysis of the *first explosive outbreak* of epidemic mortality in large American cities. As will presently appear, there was an extraordinary degree of variation amongst the different cities in respect of the initial force and duration of this first explosion. These *differences* between cities in respect of the severity and suddenness with which they were attacked by the disease constitute the first great problem which the epidemic raised. What factors had a causal influence in determining this great observed variation among cities? The full significance of this problem will be apparent when the facts of variation in force of explosive outbreak are before us. The first task of this study is to present the data in such a manner as to bring out the real extent and magnitude of the variation in the epidemic.

GENERAL SURVEY OF THE EPIDEMIC MORTALITY CURVES

In order to get in hand the general problem it is desirable to examine with some care the mortality by weeks in each of the cities dealt with. To this end figures 80 to 85 have been prepared. On these diagrams are plotted, for each city, the annual death rates per 1000 population from all causes, for each week, the data being those of the Weekly Health Index. The plotting is done on a logarithmic scale of ordinates (rates) and an arithmetic scale of abscissæ (weeks).[5] The curves begin with the week ended July 6, 1918, and continue to 1919. The scale is the same for all diagrams, though different combinations of parts of the logarithmic "decks" are used in certain cases in order to fit the diagrams to the page.

Anyone examining these curves thus collected together on a uniform scale for comparison can not fail to be impressed by the fact that there is an extraordinary amount of difference between different cities in respect of the force with which they were struck by the epidemic at its initial outbreak. Compare, for example, the Albany, Boston, Baltimore, Dayton, or Philadelphia curves with those for Atlanta, Indianapolis, Grand Rapids, Milwaukee, or Minneapolis. The former curves show an initial sudden explosive outbreak of great force, while the latter exhibit a much slower and milder increase of the mortality rate.

[5] For a discussion of the advantages of "arithlog" paper see Fisher, I., The "ratio" chart for plotting statistics, *Quarterly Publications Amer. Stat. Assoc.*, 1917, pp. 577–601, and Pearl, R., *Introduction to Medical Biometry and Statistics*, Philadelphia (W. B. Saunders Co.), 1923.

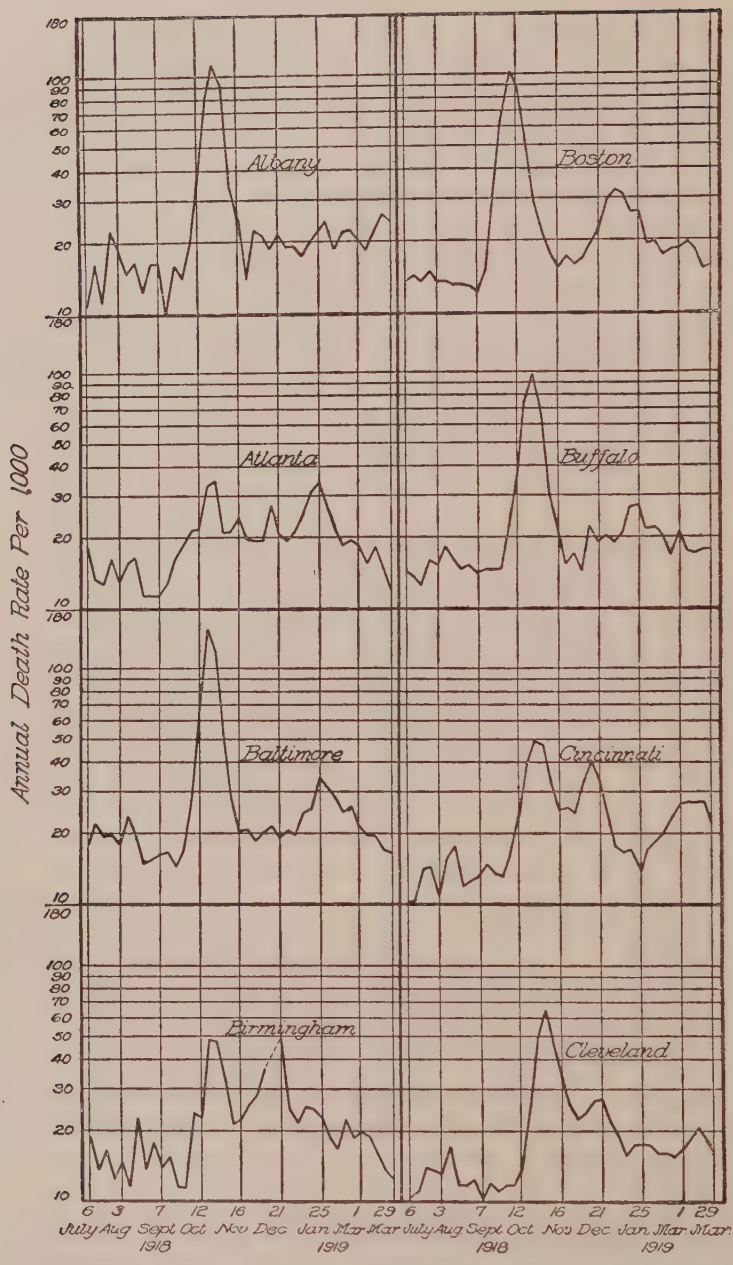

FIG. 80. ANNUAL DEATH RATES, BY WEEKS, PER 1000 POPULATION, FOR 8 CITIES

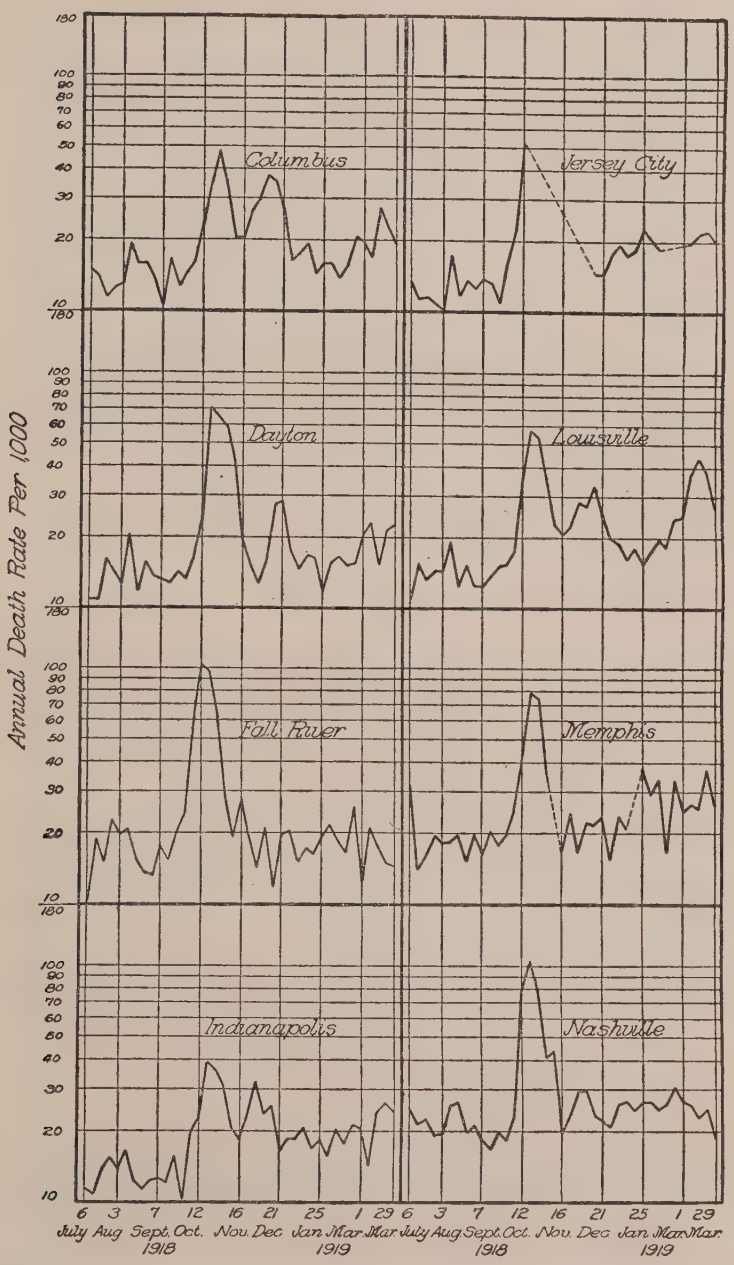
Annual Death Rate Per 1000
Columbus
Jersey City
Dayton
Louisville
Fall River
Memphis
Indianapolis
Nashville
6 3 7 12 16 21 25 1 29 6 3 7 12 16 21 25 1 29
July Aug Sept. Oct. Nov. Dec Jan Mar Mar July Aug Sept. Oct. Nov. Dec Jan Mar Mar
1918 1919 1918 1919

FIG. 81. ANNUAL DEATH RATES, BY WEEKS, PER 1000 POPULATION, FOR 8 CITIES

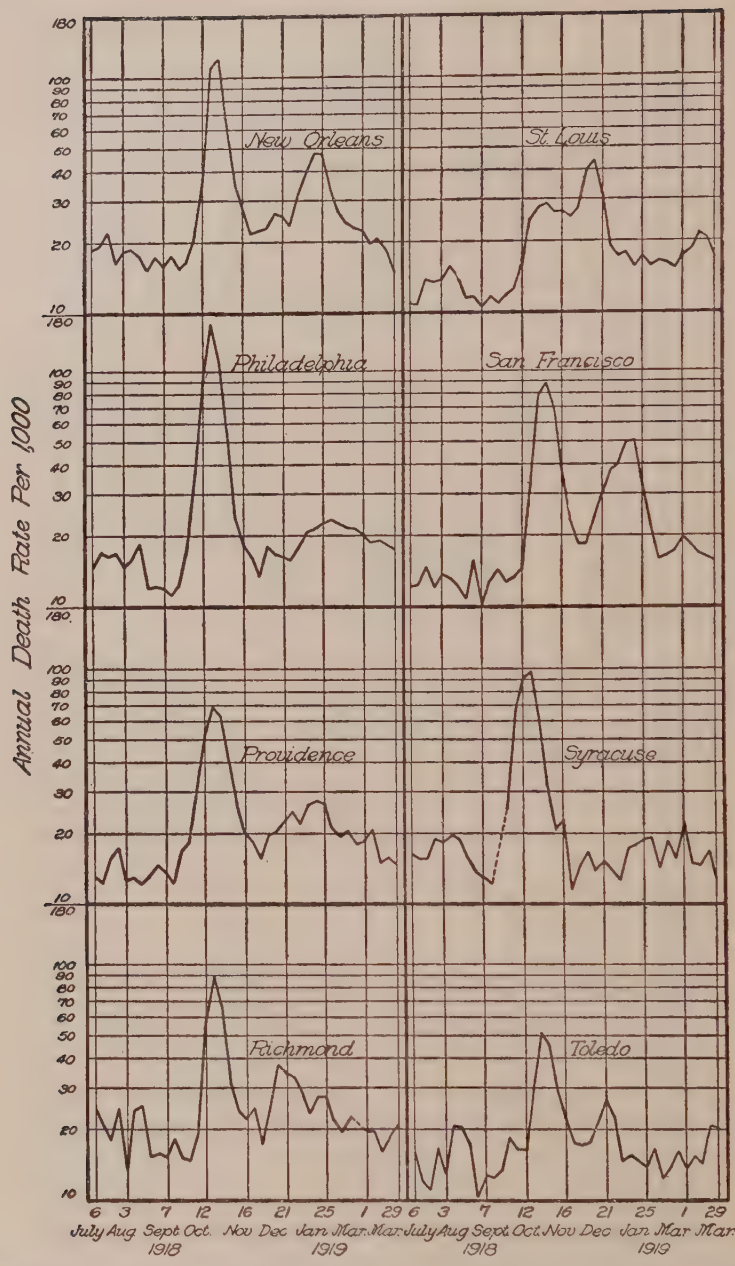

FIG. 82. ANNUAL DEATH RATES, BY WEEKS, PER 1000 POPULATION, FOR 8 CITIES

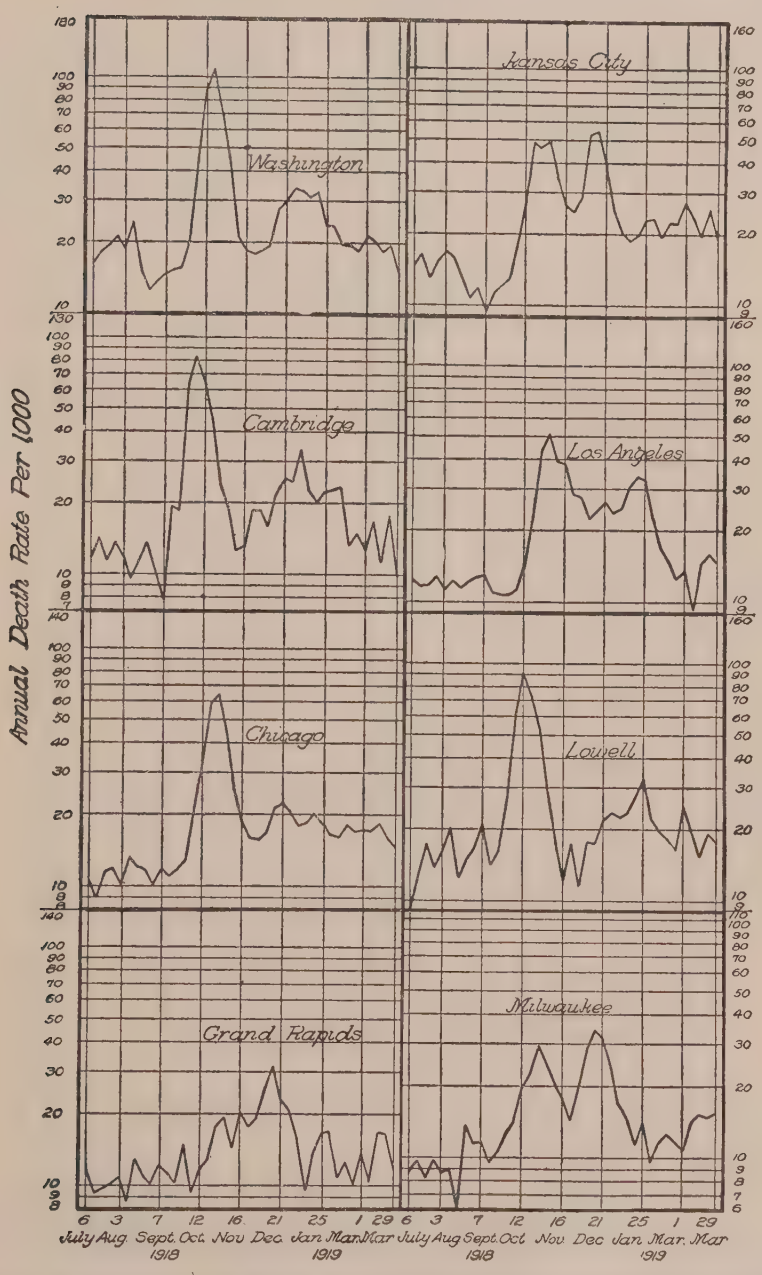
Annual Death Rate Per 1000
Washington
Kansas City
Cambridge
Los Angeles
Chicago
Lowell
Grand Rapids
Milwaukee
6 3 7 12 16 21 25 1 29 6 3 7 12 16 21 25 1 29
July Aug Sept Oct Nov Dec Jan Mar Mar July Aug Sept Oct Nov Dec Jan Mar Mar
1918 1919 1918 1919

FIG. 83. ANNUAL DEATH RATES, BY WEEKS, PER 1000 POPULATION, FOR 8 CITIES

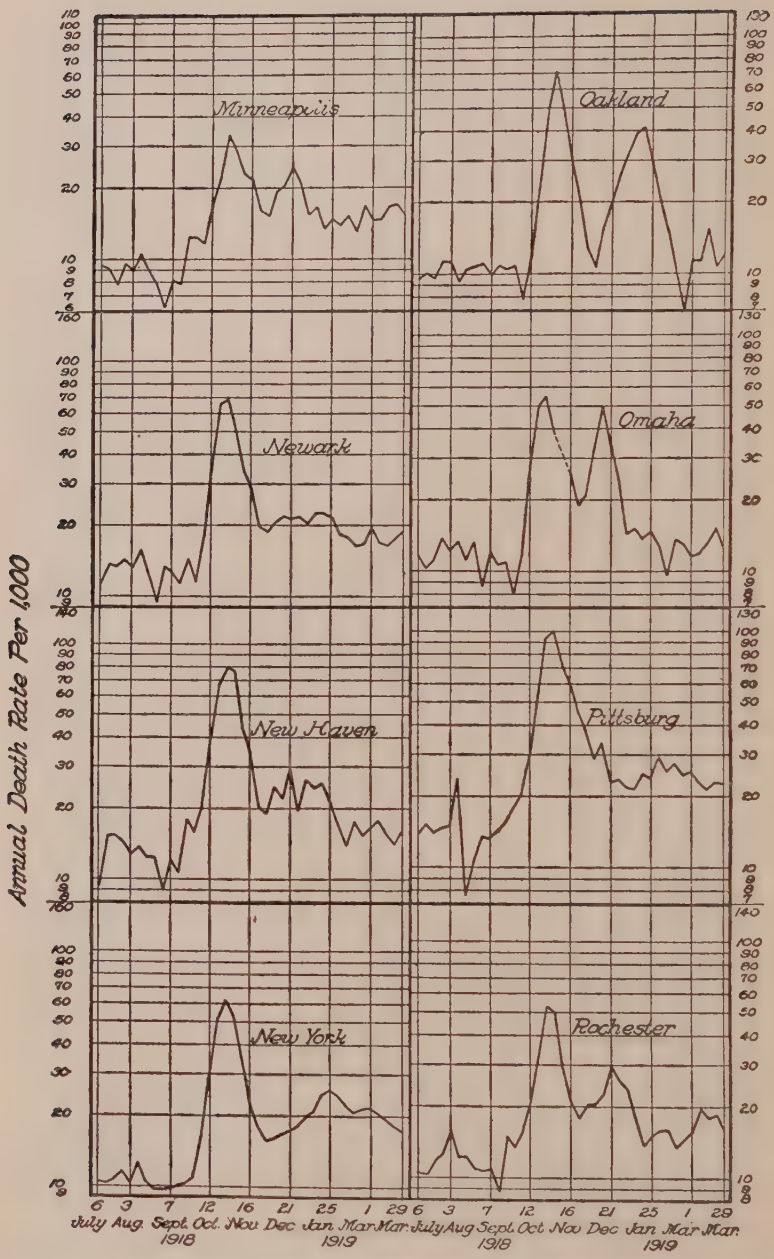
Annual Death Rate Per 1000
Minneapolis
Oakland
Newark
Omaha
New Haven
Pittsburg
New York
Rochester
6 3 7 12 16 21 25 1 29 6 3 7 12 16 21 25 1 29
July Aug. Sept. Oct. Nov Dec Jan. Mar. Mar. July Aug Sept. Oct. Nov Dec Jan. Mar. Mar.
1918 1919 1918 1919

FIG. 84. ANNUAL DEATH RATES, BY WEEKS, PER 1000 POPULATION, FOR 8 CITIES

In some cases the curve of the first epidemic outbreak rises to the peak (ascending limb) and declines from the peak (descending limb) at about the same rate. This condition of affairs is exemplified in the Albany and Baltimore curves, to mention but two. In other cases the rate of ascent to the peak is very rapid while the decline is slow and long drawn out. Such a condition is shown in the curves for Cleveland or St. Paul.

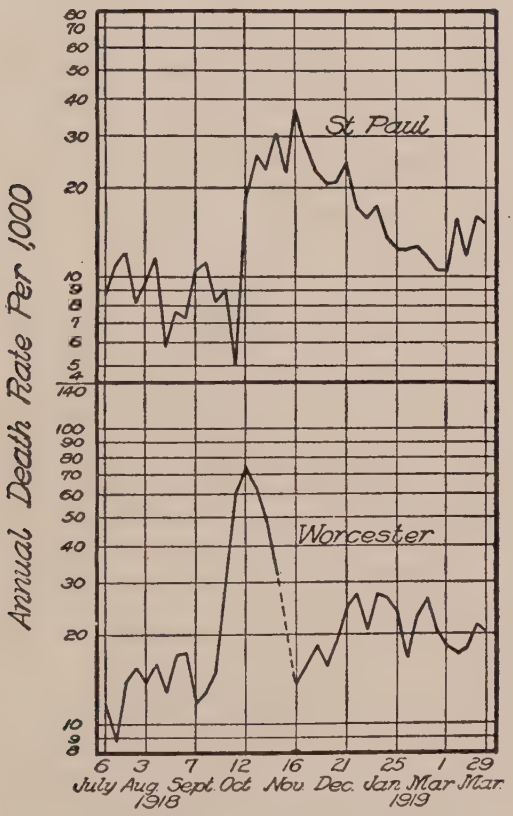

FIG. 85. ANNUAL DEATH RATES, BY WEEKS, PER 1000 POPULATION, FOR 2 CITIES

Some of the cities, such as Albany—show but a single well-defined peak in the mortality curve. Many show two peaks. Boston, New Orleans, and San Francisco give beautifully typical curves of this sort. Finally, a few of the cities show three well-marked peaks. Louisville is a good example of the latter class.

TABLE 138

Certain data regarding the time relation of the influenza epidemic in large cities

NAME OF CITY	HIGHEST PEAK RATE	REACHED ON WEEK ENDED—	NUMBER OF PEAKS	DATE OF SECOND PEAK	WEEKS, FIRST TO SECOND PEAK	DATE OF THIRD PEAK	WEEKS, SECOND TO THIRD PEAK	WEEKS, RATE WAS OUTSIDE NORMAL RANGE	WEEKS, START TO PEAK	WEEKS, PEAK TO NORMAL RATE	25-WEEK EXCESS RATE	NOTES
Albany, N. Y.	112.0	Oct. 26	1					7	3	4	4.7	
Atlanta, Ga.	35.1	Oct. 26	2	Jan. 25	13			23	4	19	2.7	A minor intermediate third peak not counted
Baltimore, Md.	148.3	Oct. 19	2	Jan. 25	14			7	3	4	6.1	
Birmingham, Ala.	48.6	Oct. 19	2	Dec. 21	9			14	3	11		
Boston, Mass.	100.2	Oct. 5	2	Jan. 4	13			9	3	6	6.5	
Buffalo, N. Y.	99.7	Oct. 26	2	Jan. 25	13			8	4	4	5.8	
Cambridge, Mass.	83.0	Oct. 5	2	Jan. 4	13			9	4	5	5.9	
Chicago, Ill.	64.2	Oct. 26	2	Dec. 21	8			8	4	4	3.8	Second peak small as compared with first
Cincinnati, Ohio	49.7	Oct. 26	3	Dec. 14	7	Mar. 22	14	17	4	13	4.0	
Cleveland, Ohio	65.6	Nov. 2	3	Dec. 21	7	Mar. 15	12	13	3	10	4.0	
Columbus, Ohio	47.2	Oct. 26	3	Dec. 7	6	Mar. 15	14	12	3	9	3.2	
Dayton, Ohio	71.6	Oct. 19	2	Dec. 21	9			8	2	6	3.5	
Fall River, Mass.	100.4	Oct. 12	1					7	3	4	5.8	
Grand Rapids, Mich.	31.6	Dec. 14	1					12	8	4	1.5	Very low, irregular curve
Indianapolis, Ind.	39.0	Oct. 19	3	Nov. 30	6	Mar. 22	16	12	3	9	2.5	Whole curve low
Louisville, Ky.	57.6	Oct. 19	3	Dec. 14	8	Mar. 15	13	14	2	12	3.6	Typical 3-peak curve
Los Angeles, Calif.	51.9	Nov. 2	2	Jan. 18	11			20	4	16	5.2	
Lowell, Mass.	89.8	Oct. 12	2	Jan. 25	15			7	3	4	5.1	
Memphis, Tenn.	79.3	Oct. 19	2	Jan. 25	14			7	3	4		
Milwaukee, Wis.	34.4	Dec. 14*	2	Dec. 14	7			16	11	5	2.9	Second peak definitely higher than first
Minneapolis, Minn.	33.1	Oct. 26	2	Dec. 21	8			22	6	16	2.7	
Nashville, Tenn.	104.2	Oct. 19	1					6	2	4	7.8	

Newark, N. J.	69.0	Oct. 26	2	Jan. 18	12			20	4	16	5.1	Both possibly 1-peaked. But 2 seems on whole best, especially for New Haven
New Haven, Conn....	78.8	Oct. 26	2	Dec. 21	8			21	6	15	5.6	
New Orleans, La.....	120.1	Oct. 26	2	Jan. 18	12			7	3	4	7.2	Beautiful typical 2-peak curve
New York, N. Y.....	61.6	Oct. 26	2	Jan. 25	13			9	4	5	4.7	
Oakland, Calif.....	70.5	Nov. 2	2	Jan. 18	11			18	3	15	5.9	It is possible that this is 3-peaked
Omaha, Nebr.....	55.1	Oct. 26	2	Dec. 14	7			15	3	12		
Philadelphia, Pa.....	158.3	Oct. 19	2	Feb. 1	15			7	3	4	7.3	Second peak low
Pittsburgh, Pa.....	100.7	Nov. 2	2	Feb. 1	13			11	4	7	8.0	
Providence, R. I.....	69.8	Oct. 19	2	Jan. 18	13			10	4	6	5.3	
Richmond, Va.....	88.4	Oct. 19	2	Dec. 14	8			5	2	3		
Rochester, N. Y.....	53.8	Oct. 26	3	Dec. 21	8	Mar. 8	11	16	4	12	2.7	Possibly this should be 2
St. Louis, Mo.....	44.0	Dec. 14†	3	Dec. 14	6	Mar. 15	13	15	10	5	3.0	
St. Paul, Minn.....	36.2	Nov. 16	1					19	6	13	3.3	Abnormal curve
San Francisco, Calif...	89.0	Nov. 2	2	Jan. 18	11			17	3	14	7.5	Typical 2-peak
Syracuse, N. Y.....	96.7	Oct. 19	1					9	4	5		
Toledo, Ohio.....	51.1	Oct. 26	3	Dec. 28	9	Mar. 22	12	6	2	4	2.1	
Washington, D. C.....	109.3	Oct. 19	2	Dec. 28	10			6	3	3	6.6	
Worcester, Mass.....	72.2	Oct. 12	2	Dec. 28	11			7	3	4		

* First peak date October 26.

† First peak date November 2.

In most cases the first peak was the highest and the second and third were progressively lower. This was not true in all cases, however. Milwaukee and St. Louis showed second peaks higher than the first. The wave-like character of the curves in general is of great interest. The usual phenomenon was a large first wave followed by a series of other smaller ones. This general characteristic of the curves is so pronounced and definite that any epidemiological theory which is to be at all adequate must take account of it.

It is evident from general inspection of these curves that there is a strong justification for taking, as the first general problem in connection with this outbreak of influenza, the significant causal factors concerned in bringing about this observed differentiation between the different cities in respect of the form of the epidemic mortality curves. The extent and definiteness of the differences between the several curves indicate that there must be discoverable clean-cut differentiating factors which influenced the influenza death rates.

CLASSIFICATION OF THE DATA

As a first step in the analysis it is desirable to make certain rough classifications of the facts brought out by the mortality curves. To this end table 138 was prepared. In this table are set forth the following data regarding each of the cities:

1. The highest peak death rate attained in any week of the epidemic up to March 29, 1919.
2. The date[6] on which the highest peak rate was reached.
3. The number of distinct peaks exhibited by the mortality curve within the time period here studied. These different peaks indicate recrudescences or waves of the epidemic.
4. The date at which the second peak in the mortality curve occurred, in the case of those cities showing 2 or more peaks.
5. The number of weeks elapsing between the first peak and the second.
6. The date at which the third definite peak, if any, occurred in the mortality curve.
7. The number of weeks elapsing between the second peak and the third.
8. The number of weeks during which the mortality rate was higher than it had been at any time between the week ended July 6, 1918, and the beginning of the epidemic. The range of fluctuation of the weekly annual

[6] It is to be understood that all dates here and throughout are as of "weeks ended" on the specified date. The original statistics are given only in weeks and hence any finer time differentiation is impossible.

death rate in the period from July to the end of September was held to be a sufficiently accurate indication of the normal range of fluctuation of the death rate in any particular city.

9. The number of weeks elapsing from the beginning of epidemic mortality to the highest peak of the curve. This gives a measure of the time factor on the ascending side of the epidemic explosion.

10. The number of weeks elapsing from the time of the highest peak of the mortality curve to the time when the curve came again within the normal range of fluctuation. This gives the time factor on the descending limb of the epidemic outbreak.

TABLE 139

Showing the frequency of occurrence of different maximum peak death rates during the epidemic

MAXIMUM PEAK RATES	NUMBER OF CITIES		
30.0– 39.9	6	10	20
40.0– 49.9	4		
50.0– 59.9	5	10	
60.0– 69.9	5		
70.0– 79.9	5	9	16
80.0– 89.9	4		
90.0– 99.9	2	7	
100.0–109.9	5		
110.0–119.9	1	2	3
120.0–129.9	1		
130.0–139.9	0	1	
140.0–149.9	1		
150.0–159.9	1		
Total	40		

11. The excess mortality rate, over the normal for the same season of the year for the same places, for the 25 weeks between September 8, 1918, and March 1, 1919. These figures were issued as a supplement to the Weekly Health Index by the Census Bureau.[7]

From this table a number of points present themselves for discussion. They may best be taken up in separate sections, in order of the successive rubrics of the table.

1. Maximum peak death rates. The highest or maximum peak rate of mortality during the epidemic varied greatly, having ranged from 31.6 in the case of Grand Rapids, Mich., to 158.3 in the case of Philadelphia.

[7] *Cf. Pub. Health Repts.*, vol. 34, no. 11, p. 505, 1919.

The distribution of the different maximum peak rates over this range is shown in detail in table 139.

From table 139 it appears that in the 40 cities considered the peak rates which were of the most frequent occurrence were, generally speaking, rates below 70. Twenty out of the 40 fell below that figure. Only 9 out of the 40

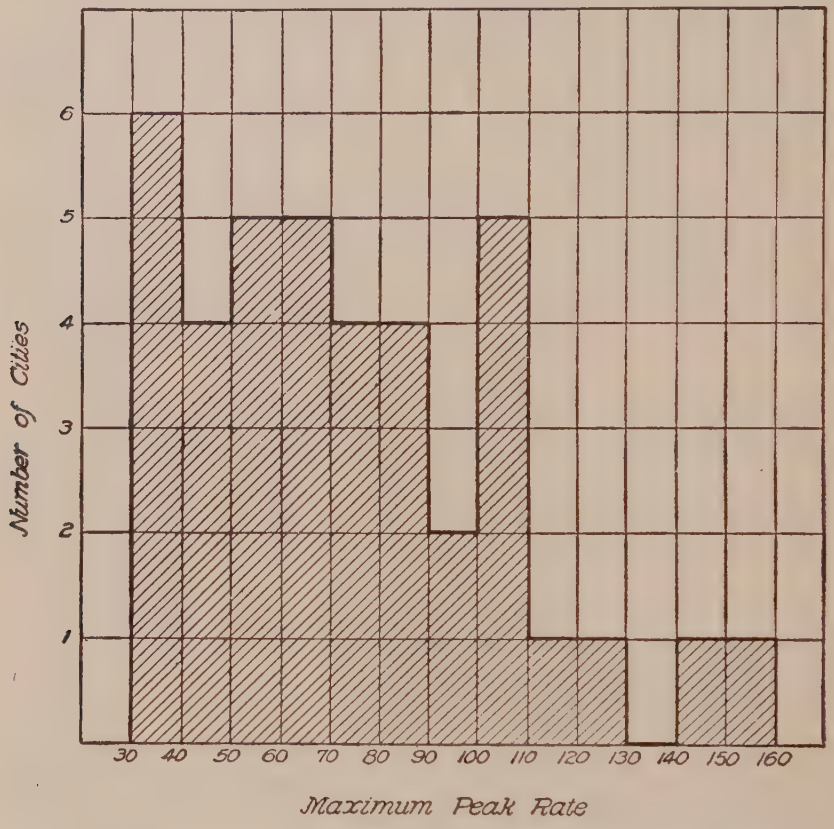

FIG. 86. DISTRIBUTION OF MAXIMUM PEAK DEATH RATES IN 40 CITIES

Certain constants of the distribution shown in table 139 are exhibited in table 140.

cities showed a maximum peak rate of 100 or more. Up to a maximum peak rate of 70 the distribution is very even in the four classes of 10 points each in the rate. From 70 on it falls off rapidly, with the single exception of the class of rate from 100 to 109.9, which has a frequency of 5.

The detailed distribution of the maximum peak rate is shown graphically in figure 86.

Two of the cities, Milwaukee and St. Louis, show higher maximum peak rates on the second wave than on the first.

2. Date of occurrence of maximum peak rate. The date of the week in which the maximum peak rate occurred is given in the third column of table 138. It will be seen that the earliest date, October 5, occurs but twice, namely, in Boston and Cambridge. These two cities, of course, are in a demographic sense practically a single unit though politically separate. At the other extreme the latest maximum peak rate date is December 14. The cities showing a culmination of the epidemic mortality during the week which ended on this latter date are Grand Rapids, Milwaukee, and St. Louis. Grand Rapids has an extremely peculiar curve, unlike that of any other city in the country. Milwaukee and St. Louis are two of the cities showing the second peak higher than the first, so in these two cases the date in the third column of table 138 refers to the second peak, while in all other cities it refers to the first peak. On these accounts the upper range end for maximum peak date should probably not be taken as December 14, but as November 2, since the

TABLE 140

Constants for maximum peak death rates

CONSTANT	VALUE
Mean maximum peak rate	73.9±3.2
Median maximum peak rate	70.0±4.0
Standard deviation	30.3±2.3

only other later date, November 16, appears in a single case, St. Paul, and the curve for that city is again abnormal. There are five cities showing the peak of the mortality curve in the week ended November 2, namely, Cleveland, Los Angeles, Oakland, Pittsburgh, and San Francisco.

The distribution of maximum peak dates is shown in table 141 and graphically in figure 87.

Using all the data, we find the following constants for date of maximum peak.

Mean peak date = October 23 ± 1.68 days.

Standard deviation in peak date = 15.75 ± 1.19 days.

These constants will serve as a useful record of the time factor in the epidemic of the autumn of 1918 in American cities.

Thirty-one out of the 40 cities had attained the peak rate of mortality prior to November 2.

3. Number of peaks in mortality curve. It is clear from the diagrams already shown that there was considerable variation in the different cities in respect of the number of epidemic mortality peaks exhibited.

TABLE 141

Distribution of dates of maximum peak mortality

MAXIMUM PEAK IN WEEK ENDED	NUMBER OF CITIES		
October 5	2	5	31
October 12	3		
October 19	12	26	
October 26	14		
November 2	5	5	6
November 9	0		
November 16	1	1	
November 23	0		
November 30	0	0	
December 7	0		
December 14	3		
Total	40		

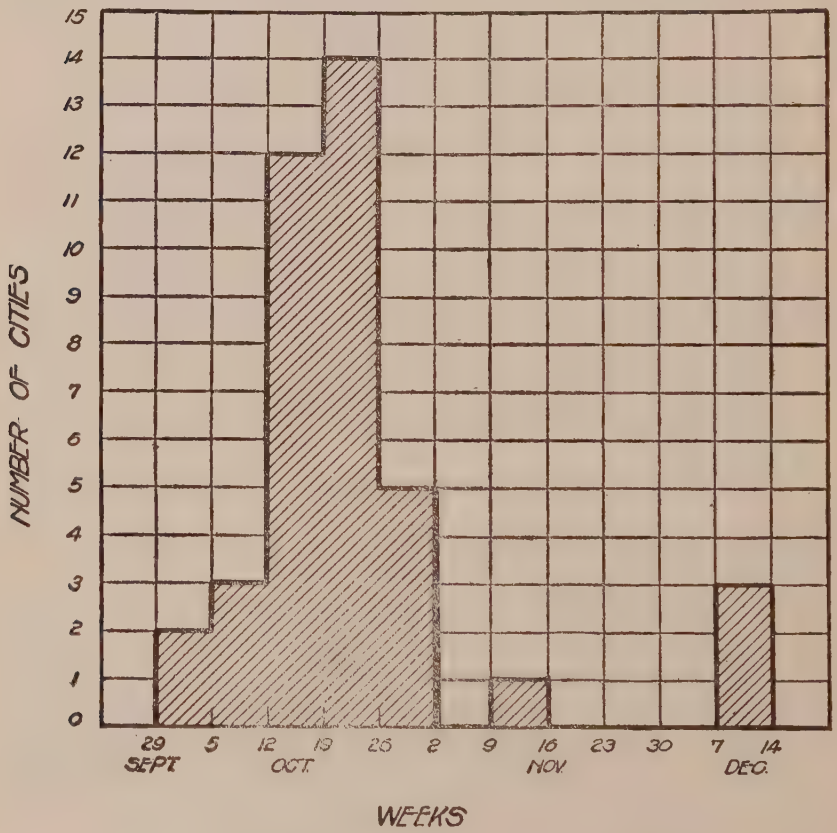

FIG. 87. DISTRIBUTION OF PEAK DATES OF THE EPIDEMIC

The details on this point are shown in table 138. Putting the data together in the form of a frequency distribution we have the results shown in table 142.

Thus it is seen that 26, or 65 per cent, of the 40 cities showed two distinct peaks in the mortality curve, while 6, or 15 per cent, had one peak, and 8, or 20 per cent, had three peaks. The diminishing wave-like character of the successive peaks is clearly shown in the diagrams.

TABLE 142

Showing number of distinct peaks in mortality curve from the beginning of the epidemic to Apr. 1, 1919.

NUMBER OF DISTINCT PEAKS	NUMBER OF CITIES	PER CENT OF CITIES
1	6	15
2	26	65
3	8	20
Total	40	100

TABLE 143

Distribution of second-peak dates

WEEK ENDED	OCCURRENCE OF SECOND PEAK IN 2-PEAK CITIES	OCCURRENCE OF SECOND PEAK IN 3-PEAK CITIES	OCCURRENCE OF SECOND PEAK IN ALL CITIES
November 30		1	1
December 7		1	1
December 14	3	3	6
December 21	5	2	7
December 28	2	1	3
January 4	2		2
January 11			
January 18	6		6
January 25	6		6
February 1	2		2
Total	26	8	34

4. Dates of second and third peaks of mortality. In the case of cities having two or three peaks the distribution of dates of occurrence of the second peak is shown in table 143.

Certain interesting facts stand out clearly from this table. In the 8 cities which had three distinct peaks of mortality the second peak came early—prior to December 28. The distribution for the 26 cities having two peaks of mortality is distinctly bimodal, 12 of them showing a mode for the week

ended December 21, and 14 a mode somewhere in the weeks of January 18 and 25. No city had a second peak of mortality in the week ended January 11.

Table 144 gives the distribution of dates of the third peak of mortality.

Here the observed mode evidently falls somewhere in the week ended March 15.

The data of tables 143 and 144 are shown graphically in figure 88.

The figures and diagram at once suggest that the group of 12 two-peak cities showing the second peak somewhere between December 7 and January 4 were cities which at that time were presumably destined to show a third distinct wave and peak of mortality, but in which for some reason not now apparent the third wave did not eventuate. In contradistinction to these stand the 14 cities showing a second peak of mortality between January 11 and February 1. These latter are presumably cities in which the complex of factors determining the form of the mortality curve was such as to lead

TABLE 144

Distribution of third peak dates

WEEK ENDED	OCCURRENCE OF THIRD PEAK
March 8	1
March 15	4
March 22	3
Total	8

definitely to a two- and only two, peaked curve. This idea will be substantiated by further evidence to be presented immediately.

As a matter of record of the epidemic in American cities, the mean dates calculated from tables 143 and 144 are given in table 145.

Putting all the data together we find for the whole group of cities the following *average* relations:

a. Days from average date of maximum peak in all cities to second peak in cities showing two or three mortality peaks = 69.26.

b. Days from date of second peak, in all cities showing two or more peaks, to third peak, in cities having three mortality peaks = 72.99.

These relations seem at first sight to point to a cycle of about ten weeks' duration in the secondary mortality waves of this influenza epidemic, after the first wave. This point can, however, be more accurately discussed by reference to the data set forth in table 138 on the number of weeks elapsing between the successive peaks.

These data are presented in the form of frequency distributions in table 146.

From this table it appears clearly that there was a definite tendency for the two-peak cities to fall into two groups in respect of the time elapsing

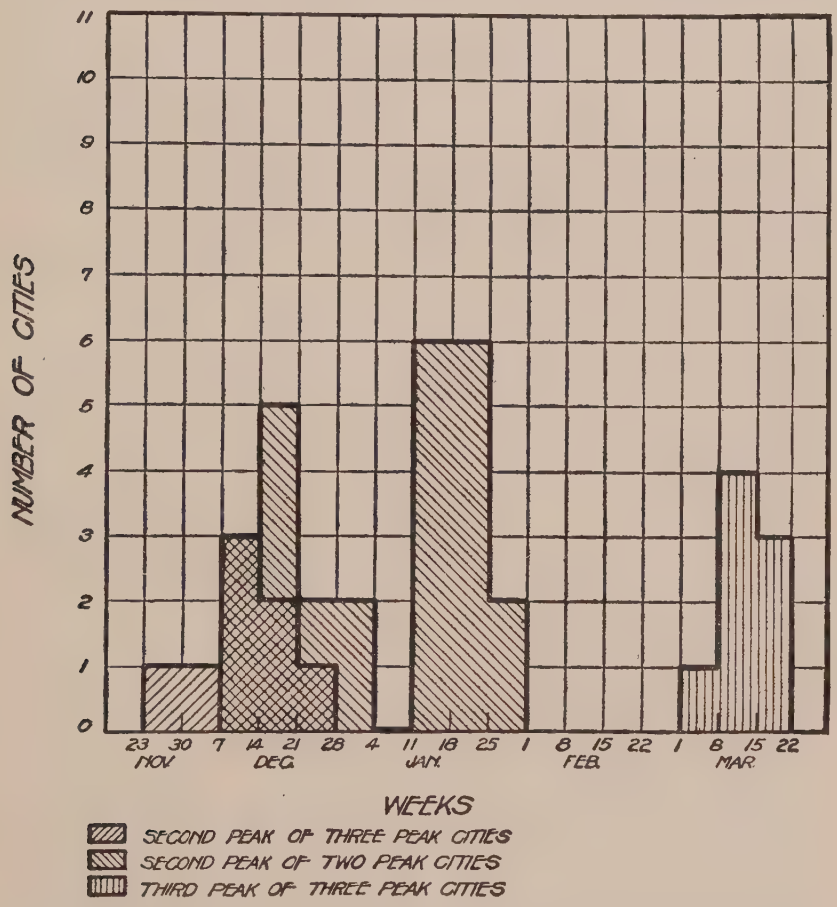

FIG. 88. FREQUENCY OF OCCURRENCE OF SECOND AND THIRD PEAKS OF MORTALITY AT DIFFERENT DATES

between first and second peaks. About a third of them had the second mortality peak around eight weeks after the first peak. The remaining two-thirds had the second peak, on the average, about thirteen weeks after the first. The three-peak curves had the second peak on an average 7.1 ±

0.3 weeks after the first, and the third peak on an average 13.1 ± 0.3 weeks after the second. The cycle in the epidemic waves would therefore appear to be nearly a multiple of seven weeks rather than the ten weeks tentatively deduced from the dates of peaks. There the process of averaging obscured the true relations.

TABLE 145

Constants for dates of second and third mortality peaks

ITEM	MEAN	STANDARD DEVIATION
Date of second peak	Jan. 1 ± 2.13 days.	18.40 ± 1.51 days.
Days from beginning of October to second peak.	92.26 days.	
Date of third peak	Mar. 14 ± 1.10 days.	4.63 ± 0.78 days.
Days from beginning of October to third peak.	165.25 days.	

TABLE 146

Frequency distributions of number of weeks elapsing between successive mortality peaks

NUMBER OF WEEKS	NUMBER OF CITIES			
	Between first and second peak			Between second and third peak
	All cities	2-peak cities	3-peak cities	
6	3		3	
7	4	2	2	
8	6	4	2	
9	3	2	1	
10	1	1		
11	4	4		1
12	2	2		2
13	7	7		2
14	2	2		2
15	2	2		
16				1
Total	34	26	8	8

5. Duration of explosive outbreak. We may next consider the question of the duration in weeks of the explosive epidemic outbreak. The pertinent data are given in the columns of table 138 headed "weeks rate was outside normal range," "weeks, start to peak," "weeks, peak to normal rate." In discussing any question of duration of an epidemic outbreak of a disease it is

necessary to define sharply and usually arbitrarily what are to be taken as limiting points. It is always difficult, and usually impossible, to define these limiting points precisely and logically so that no one will or can criticize their location. The point has recently been discussed by Hitchcock and Carey[8] who say: "The difficulty . . . . lies in deciding at just what point an undue prevalence or outbreak becomes epidemic." The general epistemological principle to be observed is clearly this: That since it is usually impossible to say with mathematical precision, in the case of an endemic disease, exactly when an epidemic outbreak begins or ends one must, in order to avoid unconscious bias in dealing with a series of different localities, lay down an arbitrary rule and follow it absolutely. Then the results will be correct *relative to each other*, even though there may be room for argument as to whether they are absolutely correct or not. Following this principle the following rule was laid down and has been used throughout: The epidemic *mortality* was considered to have begun in any city on the date when the mortality curve for that city first passed outside the range of fluctuation exhibited by the curve between the week ended July 6, 1918, and the end of the week immediately preceding the epidemic rise of the curve. The mortality of the first epidemic outbreak was considered to have ended on the date when the curve again passed within the same range of fluctuation.

This measure of duration is admittedly rough, but I think it suffices for a first approximation to the facts. It must be clearly understood that the data collected under this definition will not measure the duration of the *epidemic*, with any accuracy at all, for several reasons. In the first place, we are dealing here solely with mortality and not at all with morbidity. The mortality of an epidemic can only begin a definite and significant period of time *after* the epidemic incidence of the disease has begun. In the second place, the arbitrary definition on which we are operating here will include both peaks of some two-peaked curves and only the first peak of others, the differentiating factor being of course whether the mortality curve dropped down to within the "normal" range between peaks or did not. Now while this will seem to some a serious, not to say totally invalidating, criticism of the here defined measure of duration of first outbreak, I think it really has no weight at all. The facts are that in some cities (A) there was a sharp explosive outbreak of epidemic mortality. The death rate curve went up abruptly and came down abruptly till it was as low as it was before the epidemic outbreak. In other cities (B) the curve went up abruptly and came

[8] Hitchcock, J. S., and Carey, B. W., A median epidemic index, *Amer. Jour. Pub. Health*, vol. IX, pp. 355–357, 1919.

down, but only some part of the way, distinctly not reaching so low a rate as prevailed before the epidemic. Now by any canons of common sense it would seem clear that in the A cities the *particular epidemic outbreak* about which we are talking came to an end when the death rate was again normal for the locality and season. Subsequently the death rate may have again risen abruptly. But if it did it was a *new* and distinct epidemic outbreak, temporally and spatially related to the first outbreak if one likes, but definitely separated from it by a longer or a shorter period in which the mortality rate was *normal*. Conversely in the B cities even though the mortality rate did decline from the maximum peak rate, still it did not go back to normal, or in other words it remained an *epidemic* mortality, in the common sense of that word. The rate after this depression may have risen to a new second peak, but all the time it was part of the same epidemic outbreak. Thus it clearly appears that there is a real distinction between the A cities and the B cities. This distinction is reflected perfectly in the duration definition here adopted, and would be wholly lost in any scheme of measuring duration by peaks alone. It only needs to be kept firmly fixed in mind that we are here measuring the length of time during which the death rate was higher than the normal death rate for the same city, in the first continuous outbreak of influenza mortality.

We may first consider the total number of weeks that the mortality was outside the July to September range of fluctuation. The frequency distribution is given in table 147.

The range of variation in the duration of the first outbreak of epidemic mortality, as here defined, is great, from five weeks on the one hand (Richmond, Va.) to 23 weeks on the other (Atlanta, Ga.). So great is this variation that its general trend is not easily comprehended until the figures are somewhat combined. If that is done, certain general relations appear. First of all, it is to be noted that 20 cities, exactly one-half the total number, showed a duration as here defined of ten weeks or less, while in the other half the duration was eleven weeks or over. The median duration was then 10.5 weeks.

In general, the tendency was for the shorter durations to occur more frequently. This is well shown by figure 89, which is plotted from the last column of combined figures in table 147.

Considerably the largest single area in the histogram is the first one, covering durations of five to eight weeks inclusive. The frequencies for the longer periods, shown in four-week groups, become successively smaller.

From the ungrouped data of table 147 the following constants were calculated:

Mean duration of epidemic mortality in the first outbreak = 11.90 ± 0.55 weeks.

Standard deviation = 5.17 ± 0.39 weeks.

We may next consider the two limbs of the explosive mortality curve. The frequency distributions for the time duration of the ascending limbs and the descending limbs are given in table 148.

The first point which strikes one from this table is that it, in numerical form, confirms what is apparent from inspection of the individual curves,

TABLE 147

Frequency distribution of cities in respect of number of weeks mortality curve was outside "normal" range of fluctuation in first outbreak.

WEEKS	NUMBER OF CITIES		
5	1	4	15
6	3		
7	8	11	
8	3		
9	4	5	9
10	1		
11	1	4	
12	3		
13	1	3	7
14	2		
15	2	4	
16	2		
17	2	3	6
18	1		
19	1	3	
20	2		
21	1	2	3
22	1		
23	1		
Total	40		

namely, that (*a*) the epidemic mortality curve in the first outbreak tends in general to ascend to the peak at a more rapid rate, or in other words more abruptly than it descends; and (*b*) there is a great deal less variation among the cities in respect of the time interval covered by the ascending limb of the mortality curve than in the time required for the mortality to come from the peak rate back to normal. In 34 of the 40 cities it required four weeks or less time for the mortality rate to pass from normal to its epidemic peak. But in less than half as many (15) of the cities did the rate come down from its peak to normal again in a period of four weeks or less.

The constants of the two distributions are as follows:

Mean time from normal mortality rate to peak = 3.90 ± 0.21 weeks.

Standard deviation in time from normal mortality rate to peak = 1.93 ± 0.15 weeks.

Mean time from peak mortality rate to normal = 8.00 ± 0.50 weeks.

Standard deviation in time from peak mortality rate to normal = 4.68 ± 0.35 weeks.

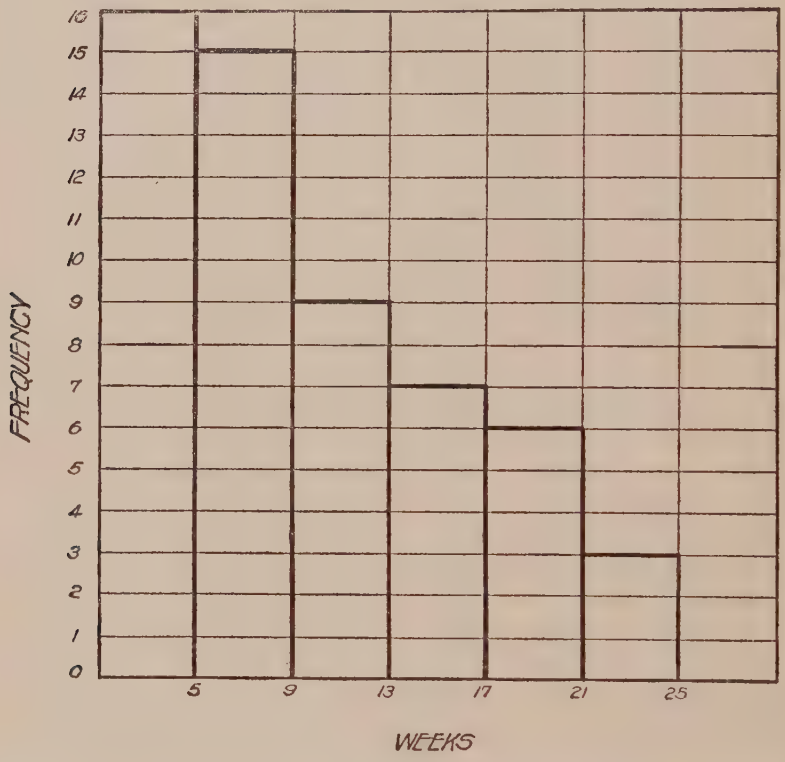

FIG. 89. FREQUENCY OF DIFFERENT DURATIONS OF THE FIRST OUTBREAK OF EPIDEMIC MORTALITY

From these figures it appears that on the average it took about twice as many weeks for the mortality curve to come back from its peak condition to the normal again, as were required for the increase from normal to peak at the beginning of the explosion. In round figures, the ascending limb of the mortality curve occupied about a month and the descending limb about two months.

TABLE 148

Frequency distributions for two moieties of epidemic mortality curve (first outbreak)

WEEKS	FREQUENCY			
	Normal to peak (ascending limb)	Cumulated frequency	Peak to normal (descending limb)	Cumulated frequency
2	5	5		
3	17	22	2	2
4	12	34	13	15
5		34	5	20
6	3	37	3	23
7		37	1	24
8	1	38		24
9		38	2	26
10	1	39	1	27
11	1	40	1	28
12		40	3	31
13		40	2	33
14		40	1	34
15		40	2	36
16		40	3	39
17		40		39
18		40		39
19		40	1	40
Total.....	40		40	

TABLE 149

Excess mortality for 25-week period

MEAN EXCESS MORTALITY RATE	NUMBER OF CITIES
1–1.9	1
2–2.9	6
3–3.9	6
4–4.9	4
5–5.9	9
6–6.9	3
7–7.9	4
8–8.9	1
Total.....	34

The differences between the two distributions of table 148 are well shown graphically in figure 90, in which the cumulated or integral curves are plotted.

6. Excess mortality. Early in March, 1919, the Census Bureau issued a supplement to its Weekly Health Index showing for 34 of the 40 cities of table 138 the mean excess rate of mortality due to the epidemic for the period of twenty-five weeks preceding March 1. These data are given in the last column of table 138. They are arranged in the form of a frequency distribution in table 149.

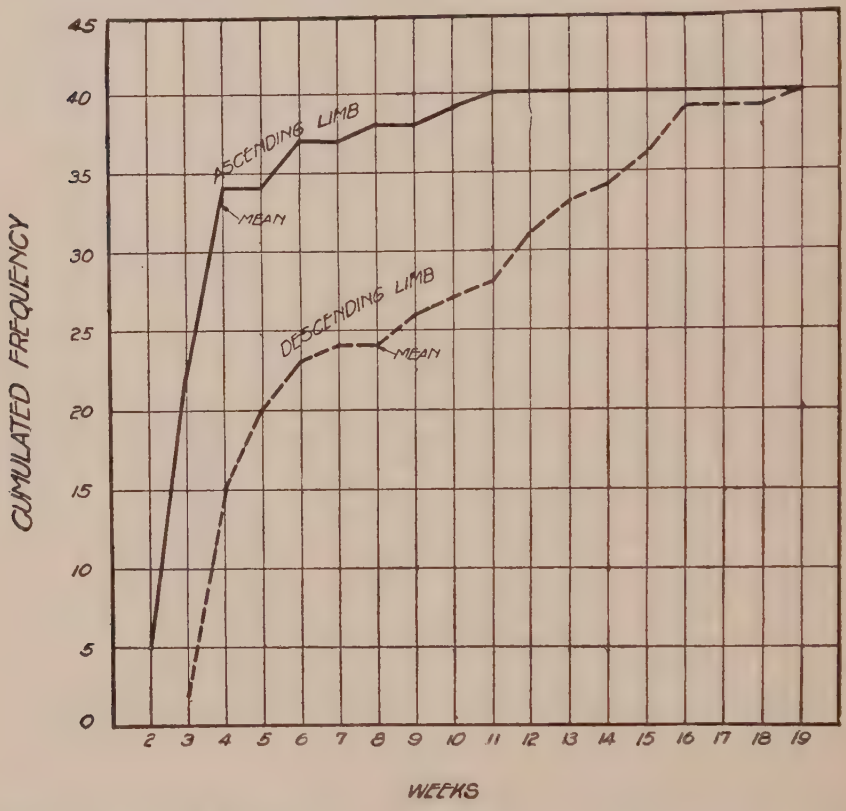

FIG. 90. CUMULATED FREQUENCY FOR TIME COVERED BY (*a*) ASCENDING LIMB AND (*b*) DESCENDING LIMB OF EPIDEMIC MORTALITY CURVES

Considering the small numbers involved, this is a fairly smooth unimodal distribution. Half of the cities have excess rates below five, and half above. Calculating from the ungrouped material we find:

Mean twenty-five-week excess mortality rate $= 4.75 \pm 0.20$.

Standard deviation in twenty-five-week excess mortality rate $= 1.76 \pm 0.14$.

7. Summary of variation data. Summarizing, it may be said that the purpose of the material so far presented is simply to place in orderly array the basic statistical characteristics of the weekly mortality curves of the 1918–1919 influenza epidemic in American cities, to the end that the extraordinarily great and entirely distinct differences between different cities in respect of the various characteristics of the epidemic may be apparent. It is essential to make this variation distinctly evident as a preliminary to the analytical discussion of its causes. It has been shown clearly that in respect of each of the following attributes or characters of the epidemic mortality there was a marked variation among the 40 American cities studied.

1. General form of mortality curve
2. Maximum peak mortality rate
3. Peak dates
4. Number of distinct peaks in mortality curve
5. Time between peaks of mortality
6. Steepness of ascending and descending limbs of mortality curve
7. Excess mortality rate
8. Duration of epidemic mortality

The variation among cities in these different epidemiological characters constitutes a problem of first-class hygienic interest and importance. Why did it exist? Why were not all cities at least reasonably alike in their influenza epidemic? If we can find sound and correct, even though only partial, answers to these questions we shall have gained greatly in that understanding of the epidemiology of influenza which must always underlie any effective control of it. It is to the analysis of this problem that attention will next be devoted.

EPIDEMICITY INDICES

With the variation data in hand one further step is necessary. We must have a single numerical measure or index of the force of the epidemic explosion in any particular place. In the earlier sections we have seen that the mortality curves in some cities have a single very sharp peak, while in other cases the curve of epidemic mortality is a long, low, flat curve. To deal practically with such differences, it is essential to have some single numerical index which will be sensitive to changes of any order in the curve, and at the same time will measure the essential characteristic which we want to measure in an epidemic curve.

Confining the discussion to mortality solely, it appears to the writer that the essential characteristic of an epidemic curve is that the death rate rises with greater or less abruptness above its normal level to a peak, more or

less pointed, and then declines again to the normal level, in a more or less steep or abrupt manner. In such a movement of the death rate curve there are two fundamental variables, namely, (*a*) the *time* during which the mortality departs from its normal level, and (*b*) the *extent* or degree of departure. If we suppose the time (*a*) made a constant then the extent of departure measures the force of epidemic mortality. In general, common sense would indicate that any measure of the force of an epidemic, or, in a single word, any measure of the epidemicity of a disease must properly incorporate both these variables.

In the discussion of the desiderata of an epidemicity index it will help to have some simple diagrams of different types of epidemics. For this purpose figures 91 and 92 are introduced. They are purely hypothetical illustrations.

In each of the two epidemics shown in these diagrams the same number of people died and the peak death rate was reached at the same time. But clearly the outbreak depicted in figure 91 would be generally regarded as a more severe or explosive epidemic, *qua* epidemic, than the one shown in figure 92. Such changes of the death rate as are shown in figure 92 may indeed not be regarded as epidemic at all. We do not commonly think of the seasonal rise in the endemic influenza rate as an epidemic. Yet it is quantitatively of the same order as the circumstances depicted in figure 92. It is of the essence of the idea of an epidemic, as commonly held, that it should have something of an explosive character—that is, there must be a relatively large increase in the death (or morbidity) rate, occurring in a relatively short space of time, in order to constitute an epidemic.

This being so, any proper measure of the degree of epidemicity must first of all measure the degree of *explosiveness* of the outbreak of the disease under discussion. There are a number of ways, mathematically, in which this can be done. The decision as to which is the best method will turn upon the degree of sensitiveness with which each measures the essentially explosive feature of the outbreak. In arriving at a measure of epidemicity for the analytical study of the influenza epidemic in American cities six different plans have been tried. The data used are the weekly mortality rates for 39 American cities dealt with in earlier sections.

1. Standard deviation of epidemic. The first epidemicity index which would occur to the biometrician is that expressed by the standard deviation of the epidemic outbreak, measured in weeks, the death rates being regarded as frequencies. An epidemic curve like that of figure 91 obviously has a smaller standard deviation in time than one such as is shown in figure 92. In general, the greater the explosiveness of the outbreak the smaller will be the standard deviation. Practically the manner in which this index is calculated is as follows:

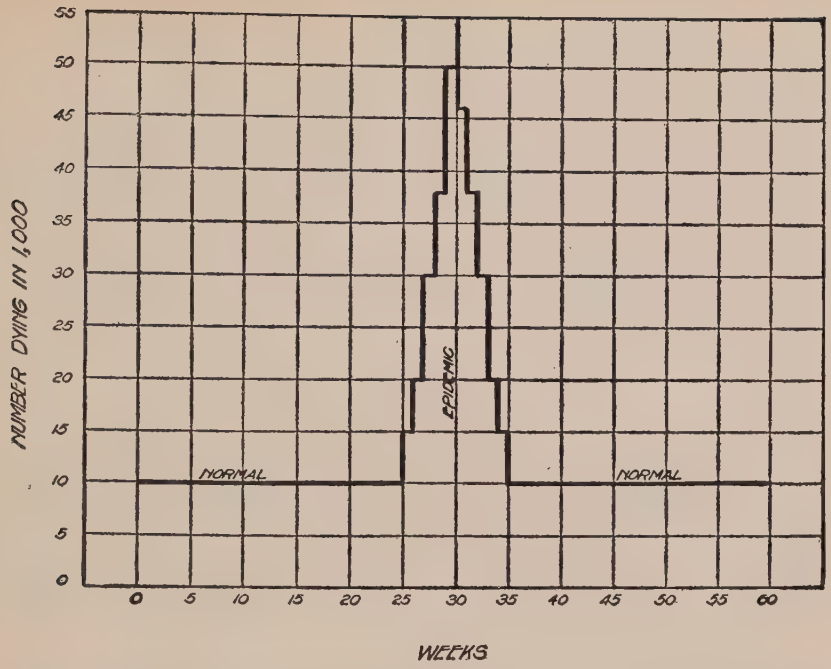

FIG. 91. HYPOTHETICAL DIAGRAM TO SHOW EPIDEMIC OF GREAT EXPLOSIVENESS

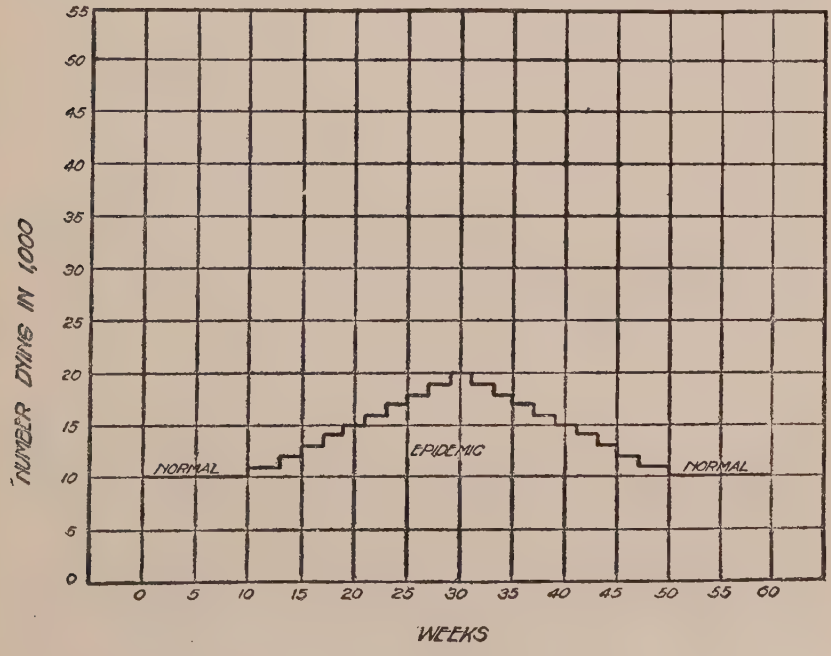

FIG. 92. HYPOTHETICAL DIAGRAM TO SHOW EPIDEMIC OF SMALL EXPLOSIVENESS

a. Take as the basis of calculation the duration of the epidemic outbreak as defined earlier.

b. Within the range so defined calculate the standard deviation in weeks in the ordinary way, the observed death rates being taken as ordinates.

In the present instance the constant takes this form: Let y denote the death rate in a particular week, and x the deviation of the week in which that rate occurred from the mean. Then, if I_1 denotes the epidemicity index, we have

$$I_1 = \sqrt{\frac{\Sigma_{x_1}^{x_n} yx^2}{\Sigma y}}$$

where Σ denotes summation. This index is easy to calculate and has a definite physical meaning. Practically, it would probably be desirable if I_1 were to be used as an epidemicity index generally, to take some multiple of its reciprocal for tabling, since as the index now stands it becomes numerically smaller as the explosiveness of the epidemic becomes greater. The value $100/I_1$ would be satisfactory.

2. Variation of excess death rates. Another measure of epidemicity which may be considered is of a more complex character than the last. Its nature may be indicated symbolically as follows:

Let M = mean death rate during epidemic, the latter being delimited as to duration by the definition in an earlier section already referred to;

M' = mean death rate in the period from July 6, 1918, to outbreak of epidemic.

$M'' = M - M'$ = increase in mean death rate during epidemic.

$S = \sqrt{\Sigma_1^n \frac{y^2}{n}}$, where y is the deviation of any particular week's death rate from M, and n is the number of weeks in the epidemic period. S is the standard deviation of the epidemic death rates, each equally weighted.

Then the second epidemicity index is

$$I_2 = \frac{100S}{M''}$$

This quantity will increase as the explosiveness of the outbreak increases. In ordinary biometric terminology it is the coefficient of variation of the weekly death rates in the epidemic period, referred to the mean excess rate as a base.

3. Mean increase in death rate during epidemic. As a third epidemicity index we may take the quantity called M'' in the preceding section. We then have

$$I_3 = M''$$

4. Twenty-five-week excess rate. It has been suggested that the average excess weekly annual death rate for the twenty-five weeks ended March 1, 1919, might be used as a measure of the force of the epidemic. Indeed, it has been so used practically by various health officials. In the present connection we may designate this measure as I_4.

5. Peak-time ratio. An epidemicity index which immediately makes strong appeal by virtue of its simplicity is a constant for any mortality curve which may be called the peak-time ratio. The symbolical expression for it is:

$$I_5 = \frac{P - M'}{T}$$

where P denotes the maximum peak mortality rate observed during the duration T of the epidemic, T being delimited by the definition stated earlier in this paper, and M' is the quantity defined under the same symbol in section 2 above. This index increases as the explosiveness of the outbreak increases.

Before discussing the sixth epidemicity index it is desirable to present quantitative data on the first five.

NUMERICAL VALUES OF EPIDEMICITY INDICES

It is evident at once that these first five indices have different degrees of validity and usefulness. Before attempting to discuss them in detail, however, it will be well to get the numerical values for each, in the case of each of the 39 cities under discussion. This is done in table 150.

Of these five indices there are only two which need to be taken seriously into account as practical working measures of epidemicity. These are the first and last, I_1 and I_5. The other three fail in that they do not adequately take account of the time or duration variable, which, as we have already seen, must be an essential factor in measuring epidemic explosiveness. These other indices really measure other aspects of the epidemic better than they do explosiveness of the outbreak, which is the thing we are just now interested in. The inadequacy of I_2, I_3, or I_4 to measure relative explosiveness of outbreak can be readily seen by comparing, city by city, the values given in these columns of table 150 with the curves for the same cities in figures 80 to 85.

As between I_1 and I_5 the advantage of I_5 is clear. It is numerically more sensitive to changes in the epidemic mortality curves. This fact is reflected in a comparison of the relative variation of the five indices which is made in table 151. For comparing the relative sensitivity of the indices to differences in the epidemic mortality curves, the ratio of the standard deviation of each index to its mean has been taken. This ratio has no significance in this case except for comparative purposes.

TABLE 150

Showing values of different epidemicity indices of mortality in American cities during influenza epidemic of 1918

CITIES	I_1	I_2	I_3	I_4	I_5
	weeks	*per cent*			
Albany	1.61	85.9	40.13	4.7	13.81
Atlanta	6.68	58.5	9.31	2.7	0.92
Baltimore	1.54	94.5	48.61	6.1	18.61
Birmingham	4.06	60.7	17.04		2.41
Boston	1.98	88.5	33.47	6.5	9.62
Buffalo	1.85	92.0	31.19	5.8	10.55
Cambridge	2.00	88.9	27.68	5.9	7.94
Chicago	1.98	72.4	24.04	3.8	6.61
Cincinnati	4.55	69.8	15.41	4.0	2.15
Cleveland	3.63	74.2	18.30	4.0	4.09
Columbus	3.55	56.4	14.94	3.2	2.74
Dayton	6.24	91.4	24.67	3.5	7.20
Fall River	1.66	80.9	38.70	5.8	11.92
Grand Rapids	3.41	65.7	8.10	1.5	1.68
Indianapolis	3.42	55.9	12.51	2.5	2.15
Louisville	4.11	78.4	15.45	3.6	3.07
Los Angeles	5.50	62.7	15.78	5.2	2.00
Lowell	1.70	71.5	34.60	5.1	10.58
Memphis	1.76	94.7	24.15		8.60
Milwaukee	4.48	57.4	11.57	2.9	1.53
Minneapolis	5.98	55.1	9.80	2.7	1.12
Nashville	1.58	72.6	39.39	7.8	13.83
Newark	5.70	99.0	15.34	5.1	2.81
New Haven	5.43	100.6	18.89	5.6	3.16
New Orleans	1.69	90.2	40.95	7.2	14.60
New York	2.19	71.2	23.29	4.7	5.67
Oakland	5.25	77.0	18.74	5.9	3.35
Omaha	4.17	69.6	18.47		2.91
Philadelphia	1.52	86.2	56.08	7.3	20.51
Pittsburgh	2.79	67.0	37.62	8.0	7.82
Providence	2.46	86.4	21.79	5.3	5.60
Richmond	1.33	66.1	35.12		13.91
Rochester	4.48	79.2	13.94	2.7	2.62
St. Louis	4.06	59.1	13.47	3.0	2.11
St. Paul	5.12	57.8	11.31	3.3	1.43
San Francisco	5.06	78.4	26.50	7.5	4.49
Syracuse	2.09	94.2	30.77		8.97
Toledo	1.67	69.8	17.19	2.1	5.95
Washington	1.49	66.3	45.08	6.6	15.34

By conventional biometric standards it might seem *a priori* that I_1 would be a better epidemicity index than I_5. Practically it is seen from table 151 that the superiority of I_5 is outstanding. The reason for this superiority appears upon analysis to be that this index relates in one of the simplest ways possible the two essential factors in relative explosiveness, namely, the height of the explosion, and the time it required, and is therefore most sensitive to differences in relative explosiveness. The same type of constant might be used for the measure of variation in frequency curves generally, except for the fact that ordinarily it is impossible to delimit the range by absolute definition, as can be done in the case of epidemics. In an ordinary frequency curve the probable error of any determination of the range is large. The nature of the definition of the range or duration which we have here adopted for epidemic curves, as well as the characteristics of epidemic curves themselves, largely reduces this probable error in the present connection. And in any case, whatever effect the probable error of the empiric determination of duration may have will tend to be greater in the case of I_1 than of I_5.

TABLE 151

Relative sensitivity of different epidemicity indices

INDEX	RATIO OF S. D. TO MEAN
I_1	0.49
I_2	0.18
I_3	0.49
I_4	0.37
I_5	0.77

THE CORRELATION OF THE EXPLOSIVENESS OF THE OUTBREAK OF MORTALITY IN THE INFLUENZA EPIDEMIC WITH VARIOUS OTHER FACTORS

We come now to the most essential part of the study, namely, the attempt to find factors directly related to or concerned in the production of the extraordinary differences between different cities in respect of the relative explosiveness of the outbreak of epidemic mortality. The method of analysis which will be followed is that of multiple correlation.[9] The general principle of the correlation method is simple. If in the present case, for example, we should find that, in general, when a city had a high influenza epidemicity index it also had a high density of population, and conversely, that cities having low epidemicity indices had low density of population, it would

[9] *Cf.* Yule, G. U., On the theory of correlation, *Jour. Roy. Stat. Soc.*, vol. LX, 1897, and On the theory of correlation for any number of variables, treated by a new system of notation, *Proc. Roy. Soc. A*, vol. 79, pp. 182–193, 1907.

be said that there was a positive correlation in variation between explosiveness of epidemic and density of population.

In a system of n variables correlation between any two, with the others remaining constant, is measured by the coefficient

$$r_{12.34\cdots n} = \frac{r_{12.34\cdots(n-1)} - r_{1n.34\cdots(n-1)} \cdot r_{2n.34\cdots(n-1)}}{(1 - r^2_{1n.34\cdots(n-1)})^{1/2}(1 - r^2_{2n.34\cdots(n-1)})^{1/2}}$$

and a coefficient of zero order is found from the observations by the following well-known expression:

$$r_{12} = \frac{S(xy)}{N\sigma_1\sigma_2}$$

In the present case, because of the statistically small number of cities for which data are available, the zero order coefficients were all determined by the direct product-moment method, without the formation of correlation tables.

The problem may be stated in this way: It is an obvious fact that the large American cities varied enormously among themselves in respect to the explosiveness of outbreak of epidemic mortality in the autumn of 1918. What factors, environmental or other, were significant in determining or influencing this variation? Or, put in another way, what factors of the numerous other respects in which these 34 large cities differ from one another can be shown to be significantly correlated with the observed differences in explosiveness of outbreak of epidemic mortality?

The variable phenomena or attributes discussed are listed below, together with the subscript numbers by which they will be designated.

SUBSCRIPT NUMBER	VARIABLE
1	Explosiveness of outbreak of epidemic mortality as measured by an epidemicity index I_6
3a	Normal death rate from pulmonary tuberculosis
3b	Normal death rate from organic diseases of the heart
3c	Normal death rate from acute nephritis and Bright's disease
3d	Normal death rate from typhoid fever
3e	Normal death rate from cancer and other malignant tumors
3f	Normal death rate from all causes
4	Age distribution of population
5	Sex ratio of population
6	Density of population
7	Latitude
8	Longitude
9	Rate of growth of population, 1900–1910

In the following paragraphs these variables are defined and discussed in detail.

1. The index, I_5, as we have seen increases as the explosiveness of the outbreak increases. It was pointed out by Dr. W. H. Frost and Mr. Edgar Sydenstricker, of the United States Public Health Service, that it was open to some criticism as a measure of explosiveness of epidemic outbreak, in the strictest sense of the term. The point of criticism was that inasmuch as T included the whole time within which the mortality curve was outside the normal range, the value of the index would be influenced by both the ascending and descending limbs of the epidemic curve; whereas if it is strict explosiveness of *outbreak* that we wish to measure, only the ascending limb is of moment. Reflection shows that the point is well taken, and consequently in the present study we have used an index I_6, for which the symbolic expression is:

$$I_6 = \frac{P - M'}{T'}$$

Here the letters have the same significance as before except that T' is the number of weeks elapsed between (*a*) the date when the mortality curve first passed outside the range of fluctuation exhibited by the curve between the week ended July 6, 1918, and the end of the week immediately preceding the epidemic rise of the curve, and (*b*) the week in which the mortality curve attained its first epidemic peak. In other words, there is now included in the epidemicity index only the ascending limb of the epidemic curve. As a matter of fact, in American cities here dealt with, little practical difference is made in any conclusions regarding the epidemic whether one uses I_5 or I_6, but there can be no question that theoretically I_6 is the superior value, and consequently we have used it.

2. The subscript 2 refers to total destructiveness of the epidemic, a variable defined in a later section.

3. The subscript 3 refers to a normal death rate in the community from one of the causes specified by letters. In the present study we have taken the mean annual death rate from each of the specified causes for the three years 1915, 1916, and 1917. It is beyond question that the three-year average will give a much more accurate representation of the prevailing normal rate of mortality from each of these diseases in the community just preceding the epidemic than will the rate for any single year.

4. Age distribution of the population. We have adopted as an age-constitution index the function

$$\phi = S\left\{\frac{\Delta^2}{P}\right\}(M - M_p)$$

which is discussed in Chapter XVI of this book.

5. As an index of the sex distribution of the population, the male sex ratio was expressed as the ratio of males to 100 females in 1910.

6. The density of population in each city was calculated from data furnished in the *Financial Statistics of Cities*, issued annually by the Bureau of the Census, and was expressed as the number of persons per acre of land area within the legally defined limits of the city.

7 and 8. The interest in having some measure, in a study of this kind, of geographical position is twofold: First, that which arises from purely epidemiological considerations, namely, as affording, in relation to time, an index of the rate of spread of an infectious epidemic disease from a primary focus; and secondly, the fact that geographical position, especially latitude, is a rough but on the whole fairly accurate index of general climatological conditions. It was decided to make the expression of geographical position as accurate as possible in the present study, and consequently there were included as definite variables the latitude and longitude of each of the cities considered.

9. Rate of growth of population 1900–1910. The reason for the inclusion of this variable in the study was twofold. Primarily this may be taken as a rough but probably fairly accurate index of the degree of industrialization of a city. In general, those cities which are growing most rapidly in population are those in which the most rapid industrial development is taking place. It would be much better if the rate of growth of the population between 1910 and the outbreak of the epidemic could have been used; but accurate data are lacking, nor will they be available until the results of the 1920 census are published. Consequently this variable must be regarded as a rough approximation to one that we should like to measure more accurately, namely, the present state and recent rate of industrial development. In the second place, rate of growth is a definite biological characteristic of a population,[10] and as such worthy of inclusion in any study relating an epidemic disease to demographic conditions.

DATA

The actual data used in the correlations are given in table 152. Since in other work it was desired to correlate destructiveness of the epidemic, as measured by the twenty-five-week excess mortality, with the other variables,

[10] *Cf.* Pearl, R., and Reed, L. J., On the rate of growth of the population of the United States since 1790 and its mathematical representation, *Proc. Nat. Acad. Sci.*, vol. 6, pp. 275–288, 1920; also Chapter XXIV of this book.

only 34 cities could be used, because only for that number are the excess mortality figures available.

DEMOGRAPHIC AND ENVIRONMENTAL CORRELATIONS

We come now to the consideration of the results. The net influence of the several demographic and environmental factors upon explosiveness of epidemic outbreak may be first considered. As we are obviously interested in getting at the *net* influence of each factor, such as age distribution of the population, upon variation in the epidemicity index, while all the other factors for which we have data are held to constant values, we may pass at once to the fifth order correlation coefficients, without stopping for detailed consideration of the lower order coefficients leading to the final values. Such points as do need discussion in connection with these lower order coefficients will be brought out in connection with the fifth order results. The net correlations between epidemicity index (subscript 1) and each of the six demographic and environmental factors taken one at a time, with the other five held constant, are exhibited in table 153. It is to be understood that subscript numbers to the right of the decimal point denote variables held constant, and subscript numbers to the left of the point denote the variables correlated. r is, of course, simply the conventional symbol of a correlation coefficient.

Taking the several variables in order we note:

1. Explosiveness of outbreak of epidemic mortality can not be positively asserted to be significantly correlated with the age distribution of the population. The coefficient $+ 0.281$ is less than 3 times its probable error. The plus sign means, having regard to the method of calculating age indices explained above, that so far as there is any correlation, high values of the explosiveness index I_6 tended to be associated with populations having a higher proportion of *older* persons than the average. No one of the lower order correlations of explosiveness and age index had a value as much as 3 times its probable error. The highest coefficient in the series was the fourth order $r_{14 \cdot 6789} = + 0.300 \pm 0.105$. The zero order $r_{14} = +0.194 \pm 0.111$. All r_{14} correlations, whatever the secondary subscripts, within the group now under consideration, are positive. With border line values such as these, one can only say that in cities constant in respect to sex ratio of population, density of population, position, and rate of recent growth of population, the age composition of the population may have a slight influence in determining explosiveness of outbreak of epidemic mortality, but at best the influence must be very small.

TABLE 152

Data for correlation of characteristics of cities with explosiveness of epidemic influenza mortality.

(The subscript numbers at the heads of columns correspond to the list of variables given on p. 474.)

CITY	1	3a	3b	3c	3d	3e	3f	4	5	6	7	8	9
Albany, N. Y.	32.2	224.5	236.4	180.1	10.8	137.6	1,947.0	−10.73	92.9	8.89	42.65	73.75	6.5
Atlanta, Ga.	5.3	113.9	121.6	159.2	19.5	66.5	1,551.2	−82.71	92.7	11.42	33.73	84.33	72.3
Baltimore, Md.	43.4	202.7	195.9	179.0	18.1	107.5	1,810.3	−31.80	92.4	30.57	39.28	76.62	9.7
Boston, Mass.	28.8	143.8	211.5	101.0	3.9	117.0	1,651.6	−31.05	96.7	27.36	42.36	71.06	19.6
Buffalo, N. Y.	21.1	146.0	165.9	126.0	10.3	97.8	1,566.4	−48.61	100.6	18.97	42.88	78.92	20.2
Cambridge, Mass.	17.9	178.8	183.9	73.6	2.7	118.8	1,354.4	−30.26	91.7	28.23	42.38	71.13	14.1
Chicago, Ill.	13.2	139.5	159.1	110.4	4.1	88.8	1,457.1	−68.76	106.3	20.28	41.88	87.60	28.7
Cincinnati, Ohio	9.2	206.8	202.5	162.0	4.8	111.5	1,618.0	−22.93	95.4	9.10	38.14	84.42	11.6
Cleveland, Ohio	17.7	132.8	118.8	90.0	7.0	82.4	1,462.1	−74.51	106.6	20.08	41.50	81.70	46.9
Columbus, Ohio	11.0	131.8	155.2	88.0	11.4	105.1	1,498.3	−29.08	101.5	15.18	40.00	83.00	44.6
Dayton, Ohio	28.8	139.2	184.3	106.2	17.3	107.7	1,492.7	−24.10	101.9	12.65	39.73	84.18	36.6
Fall River, Mass.	27.8	139.6	164 0	108.5	14.3	83.9	1,639.4	−73.39	93.4	5.91	41.70	71.15	13.8
Grand Rapids, Mich.	2.5	76.3	146 4	94.7	19.0	98.4	1,258.8	−27.95	97.4	11.85	42.97	85.70	28.6
Indianapolis, Ind.	8.6	164.2	185.7	111.1	17.1	95.3	1,550.2	−24.23	98.7	10.96	39.67	86.13	38.1
Louisville, Ky.	21.5	160.1	164.7	160.1	14.2	83.5	1,542.1	−32.19	94.1	16.61	38.20	85.70	9.4
Los Angeles, Calif.	10 0	182.1	156.0	106.8	4.7	103.5	1,238.2	−13.61	103.9	2.40	34.08	118.20	211.5
Lowell, Mass.	24.7	112.5	157 7	90.7	11.2	79.8	1,681.9	−34.91	94.1	13.63	42.65	71.32	11.9
Milwaukee, Wis.	4.9	82.4	100.7	76.7	8.8	85.3	1,214.4	−62.17	102.8	26.92	43.05	87.95	31.0
Minneapolis, Minn.	4.1	120.4	114.1	101.3	6.5	93.8	1,189.4	−55.39	109.2	11.27	44.97	93.30	48.7
Nashville, Tenn.	41.5	189.3	198.7	126.1	30.0	83.3	1,683.7	−48.32	89.6	10.11	36.15	86.80	36.5
Newark, N. J.	14.1	151.0	148.3	137.1	4.3	88.5	1,412.2	−62.54	99.6	27.52	40.75	74.17	41.2
New Haven, Conn.	11.1	103.6	185.4	125.1	13.0	112.1	1,660.0	−33.70	99.7	13.06	41.32	72.92	23.7
New Orleans, La.	34.1	270.9	224.4	245.6	22.3	95.5	1,983.0	−52.44	92.8	2.96	29.97	90.08	18.1
New York, N. Y.	12.8	160.5	165.3	128.3	4.6	85.0	1,384.7	−74.42	99.9	29.54	40.71	74.00	38.7
Oakland, Calif.	15.1	98.7	187.6	87.7	4.4	94.2	1,082.8	−17.18	108.7	6.41	37.75	122.33	124.3

Philadelphia, Pa.....	47.9	169.9	203.5	178.3	6.9	97.6	1,630.1	−34.27	96.4	21.02	39.95	75.17	19.7
Pittsburgh, Pa.....	21.3	115.9	134.5	94.3	10.4	90.7	1,694.0	−71.77	105.1	22.81	40.43	80.03	18.2
Providence, R. I.....	14.0	133.3	152.8	130.2	6.4	99.1	1,527.0	−30.05	96.7	22.35	41.84	71.40	27.8
Rochester, N. Y.....	13.9	95.7	203.1	138.0	4.7	109.8	1,456.8	−26.62	98.7	18.62	43.13	77.85	34.2
St. Louis, Mo.....	5.4	132.2	141.3	171.9	8.1	97.3	1,458.0	−44.57	101.5	19.36	38.63	90.20	19.4
St. Paul, Minn.....	4.5	106.6	118.9	79.7	5.0	86.2	1,093.0	−68.95	108.6	7.40	44.87	93.08	31.7
San Francisco, Calif.....	25.4	170.0	237.2	132.5	5.7	138.0	1,546.5	−34.34	131.6	17.55	37.79	122.43	21.6
Toledo, Ohio.....	17.9	173.7	185.2	88.7	18.0	103.9	1,705.1	−33.21	101.1	10.91	41.77	83.55	27.8
Washington, D. C.....	30.7	185.5	235.1	176.2	12.7	110.5	1,795.6	−20.67	91.3	9.55	38.89	77.03	18.8

2. The sex ratio of the population is plainly not significantly correlated with epidemicity index. The fifth order coefficient has a value -0.001 ± 0.116, which is sensibly zero. This is an interesting example of how partial or net correlations may differ from total correlations. The zero order coefficient between explosiveness (I_6) and sex ratio is $r_{15} = -0.307 \pm 0.105$. This is a probably significant value, but arises not from any direct relation of variables 1 and 5, but indirectly through the relation of both of these to the positional variables 7 and 8 (latitude and longitude). In the group of cities here dealt with there is a relatively high correlation between male sex ratio and longitude, and between sex ratio and latitude, when longitude is constant. These relations are shown in the following coefficients:

$r_{57} = +0.134 \pm 0.114$ (sex ratio and latitude)
$r_{58} = +0.678 \pm 0.062$ (sex ratio and longitude)
$r_{57 \cdot 8} = +0.607 \pm 0.073$ (sex ratio and latitude—longitude constant)
$r_{58 \cdot 7} = +0.808 \pm 0.040$ (sex ratio and longitude—latitude constant)

TABLE 153

Net correlation of explosiveness of outbreak (I_6) with various demographic and environmental factors

VARIABLE CORRELATED WITH EXPLOSIVENESS (I_6)	r SUBSCRIPTS	COEFFICIENT
Age distribution of population	14.56789	$+0.281 \pm 0.107$
Sex ratio of population	15.46789	-0.001 ± 0.116
Density of population	16.45789	$+0.099 \pm 0.115$
Latitude of city	17.45689	-0.369 ± 0.100
Longitude of city	18.45679	-0.085 ± 0.115
Rate of growth of population, 1900–1910	19.45678	-0.288 ± 0.106

Because of the inverse and nearly equal correlations of epidemicity index with latitude and longitude, the sex-ratio correlation with epidemicity index is neutralized as soon as these other variables are brought into the system. Thus while we have $r_{15} = -0.307 \pm 0.105$, we get $r_{15 \cdot 78} = +0.023 \pm 0.116$, or practically zero. We may then safely conclude that the proportion of males (or of females) in the population of a city had no sensible direct influence in determining the explosiveness of outbreak of epidemic mortality.

3. The net fifth order correlation of explosiveness of outbreak of the epidemic mortality with density of population is again sensibly zero. This is true whatever the variables held constant within the group here discussed. The zero order coefficient is $r_{16} = +0.073 \pm 0.115$. Nowhere in the series does a coefficient having primary subscripts 16 rise to a value even approaching 3 times the probable error. This result, that density of population, which

is the measure of urban crowding in this case, had nothing to do with determining the explosiveness of outbreak of the epidemic mortality, while surprising on grounds of purely *a priori* logic—always, by the way, most unsafe grounds—seems to be firmly established.

4. In the case of the correlation of explosiveness of outbreak of epidemic mortality with the latitude of the city, a very different result appears. The fifth order coefficient is $r_{17\cdot45689} = -0.369 \pm 0.100$. This is more than three times its probable error and is probably *statistically* significant; but

FIG. 93. OUTLINE MAP OF THE UNITED STATES SHOWING THE LOCATION OF THE 34 CITIES USED IN THIS STUDY

before drawing any conclusions about its epidemiological significance we must critically look into its genesis. In the first place it must be noted that the 34 cities with which we are dealing are not scattered at random over the United States. All but three of them are either on or east of the Mississippi River. This distribution is well shown in the map exhibited as figure 93.

Not only are the cities mostly in the eastern half of the country, but, what is more important, they nearly all fall in a fairly narrow northeast-southwest belt. How clearly this is so is shown by the regression of latitude on longitude, which is plotted on the map.[11] The distribution of these cities is

[11] It should be noted that this regression is linear. The curvature of the regression line in figure 93 is merely a recognition of the fact that the projection of the map itself is such as to show latitude lines curved.

so far from random over the whole area of the country that there is a statistically significant correlation for these 34 cities, between latitude and longitude, the coefficient being $r_{78} = -0.404 \pm 0.097$. In words, what this means is that, within this group of 34 cities, in general, the farther north a city is the farther east it is.

Now, neither latitude nor longitude is alone significantly correlated with the epidemicity index I_6, as witness the zero order coefficients

$$r_{17} = -0.243 \pm 0.109$$
$$r_{18} = -0.229 \pm 0.110$$

But because of the relatively large value of r_{78} which has just been pointed out we get at once

$$r_{17 \cdot 8} = -0.376 \pm 0.099 \text{ (significant)}$$
$$r_{18 \cdot 7} = -0.369 \pm 0.100 \text{ (significant)}$$

The same influence makes itself felt throughout the series of ascending order partial coefficients. Thus, the relevant second order coefficients are:

$r_{17 \cdot 45} = -0.208 \pm 0.111$
$r_{17 \cdot 46} = -0.303 \pm 0.105$
$r_{17 \cdot 48} = -0.387 \pm 0.098$
$r_{17 \cdot 49} = -0.407 \pm 0.097$
$r_{17 \cdot 56} = -0.264 \pm 0.108$
$r_{17 \cdot 58} = -0.319 \pm 0.104$
$r_{17 \cdot 59} = -0.358 \pm 0.101$
$r_{17 \cdot 68} = -0.381 \pm 0.099$
$r_{17 \cdot 69} = -0.403 \pm 0.097$
$r_{17 \cdot 89} = -0.421 \pm 0.095$

$r_{18 \cdot 45} = -0.092 \pm 0.115$
$r_{18 \cdot 46} = -0.242 \pm 0.109$
$r_{18 \cdot 47} = -0.407 \pm 0.097$
$r_{18 \cdot 49} = -0.074 \pm 0.115$
$r_{18 \cdot 56} = +0.022 \pm 0.116$
$r_{18 \cdot 57} = -0.245 \pm 0.109$
$r_{18 \cdot 59} = +0.194 \pm 0.111$
$r_{18 \cdot 67} = -0.333 \pm 0.103$
$r_{18 \cdot 69} = -0.061 \pm 0.115$
$r_{18 \cdot 79} = -0.160 \pm 0.113$

It is at once evident that most of the r_{17} or latitude coefficients are three or more times their probable errors. Most of the r_{18}, or longitude coefficients are, on the contrary, less than three times their probable errors, the only ones arising to a higher value being those carrying 7 as a secondary subscript and not all of those. In other words, we come here to a separating point between the correlations carrying 1 and 7 as primary subscripts and those carrying 1 and 8 in the same position. This divergence comes about from the different correlations of certain of the other variables with latitude and longitude. Thus we have, for sex-ratio correlations, $r_{75} = +0.134 \pm 0.114$ against $r_{85} = +0.678 \pm 0.062$. Age distribution index is not significantly correlated with either latitude or longitude in this group of cities. Density is significantly and about equally correlated with both, the coefficients being $r_{86} = -0.424 \pm 0.095$, and $r_{76} = -0.371 \pm 0.100$. Rate of growth in this

group of cities is nearly twice as highly correlated with longitude as with latitude, the signs of course being opposite. The coefficients are $r_{79} = -0.365 \pm 0.100$ and $r_{89} = +0.642 \pm 0.068$.

Without pursuing this complex trail further, certain things are clear. In the first place it is evident that it will be quite unsafe to draw biostatistical or demographic conclusions about, or on the basis of data from, the large American cities, without having critical regard for the fact here demonstrated that some of the most important of these characteristics are significantly correlated with the mere geographical position of the cities. In the second place, because of this fact, we can not be quite sure of the epidemiological significance of the fact that, in this group of cities, there is a statistically significant negative correlation between epidemicity index and latitude. Taken at its face value this coefficient means that, on the average, the outbreak of epidemic mortality was *more* suddenly explosive, the farther *south* the city, when the other factors of age distribution, sex ratio, density, and rate of growth of population were constant and equal. Whether this bespeaks a real and general biological phenomenon resting presumably upon a climatological base can not be critically determined until we can study the matter in a group of localities distributed in a more random manner than in the present sample, so that the correlations between latitude and longitude shall be more nearly zero in value. It is interesting to note, however, that the present result in respect to latitude correlation is in accord with general clinical and pathological experience. I am told by Dr. William H. Welch that it has long been recognized by clinicians and pathologists that acute respiratory infections, and particularly the pneumonias, tend to become more fatal as one passes from north to south. There is a splendid chance here for a critical statistical investigation of the matter, and with the rapid extension of the registration area into the South in recent years, adequate data will shortly be available.

5. Rate of growth of population of these cities in the decade 1900 to 1910 is a factor connected with age of city, with its industrial and commercial prosperity and activity, and indirectly with sanitation, because usually in cities growing very rapidly, sanitary arrangements tend to lag behind the need for them. The net fifth order correlation of this variable with explosiveness of outbreak of epidemic mortality is $r_{19 \cdot 45678} = -0.288 \pm 0.106$. This is a border-line value, which can not be safely asserted to differ significantly from zero. Roughly speaking, a value as large as this would turn up purely by chance about 7 times in every 100 trials with samples of the size here dealt with. The gross, zero order coefficient between these variables is $r_{19} = -0.302 \pm 0.105$, again not a significant value.

No one of the partial coefficients having 1 and 9 as primary subscripts is certainly significant in comparison with its probable error, except such as carry also 7 as a secondary subscript. On the whole, it can be safely asserted that if the rate at which a city had recently been growing in population had anything at all to do with the degree of explosiveness of outbreak of the 1918 epidemic mortality, this influence must have been at the most extremely slight.

6. In general, the result of this critical study of the influence of these demographic and environmental factors is to show that the only variable in the lot that has a statistically significant net correlation with the explosiveness index is latitude. I have already indicated fully the wisdom of caution for the present in drawing biological conclusions from this latitude correlation. It is possible that the age distribution of the population and the rate of recent growth of the city had some slight influence upon the explosiveness of outbreak of the epidemic mortality; but in either case the effect must have been so slight as to be negligible so far as any practical epidemiological significance is concerned.

TABLE 154

Net correlation of explosiveness of outbreak (I_6) with the normal death rates from certain specified causes

VARIABLE CORRELATED WITH EXPLOSIVENESS (I_6) DEATH RATE FROM:	r SUBSCRIPTS	COEFFICIENT
All causes	13f. 456789	+0.572±0.078
Pulmonary tuberculosis	13a. 456789	+0.389±0.098
Organic diseases of the heart	13b. 456789	+0.562±0.079
Acute nephritis and Bright's disease	13c. 456789	+0.307±0.105
Typhoid fever	13d. 456789	+0.105±0.114
Cancer and other malignant tumors	13e. 456789	+0.141±0.113

DEATH RATE CORRELATIONS

We may turn now to the correlations of explosiveness of outbreak of epidemic mortality with the normal death rates from certain primarily organic diseases. As has already been pointed out, we have in the present study taken the mean death rate for three years as indicative of normal conditions instead of a single year.

The sixth order coefficients are exhibited in table 154. These coefficients measure the net correlation existing between the epidemicity index I_6 and the specified normal death rate, when the cities involved have been made constant and equal in respect to age and sex constitution of population,

to density and rate of recent growth of population, and to latitude and longitude.

It is at once evident that these correlations are of a generally different order of magnitude from those of table 153. Specifically we note:

1. The highest correlation is that for the death rate from all causes; but that for the organic diseases of the heart is practically identical. These coefficients are more than seven times the probable error and are certainly significant. The corresponding zero order coefficients are:

All causes, $r_{13f} = +0.678 \pm 0.063$
Organic heart, $r_{13b} = +0.642 \pm 0.068$

From these values, in comparison with those of table 154, it appears that by making all six demographic and environmental factors constant, the correlation between explosiveness and the normal death rate from all causes or that from organic diseases of the heart is not altered to a degree statistically significant. This comparison indicates the overwhelming importance of the biological factor which these death rates measure in determining the explosiveness of the outbreak of epidemic mortality, as compared with the demographic and environmental factors previously considered.

2. Next to organic diseases of the heart, pulmonary tuberculosis is the single cause having its normal death rate most highly correlated with explosiveness of outbreak of the epidemic. The net sixth order coefficient for this disease, however, is distinctly lower than that for organic diseases of the heart. Furthermore it is much more reduced by the process of making demographic and environmental factors constant, as is indicated by the fact that the zero order coefficient for this disease is $r_{13a} = +0.578 \pm 0.077$. The difference between this and the sixth order coefficient $r_{13a \cdot 456789} = +0.389 \pm 0.098$ is 0.189 ± 0.125. While not statistically significant in comparison with the probable error, it comes much nearer being so than the corresponding difference for the organic heart correlation, which is 0.080 ± 0.104.

3. In the case of normal death rate from breakdown of the kidneys (acute nephritis and Bright's disease) the sixth order net coefficient is on the border line of probable statistical significance, having a value just under three times its probable error. Again the process of making the six demographic and environmental factors constant has materially reduced the correlation between the normal death rate from these diseases of the kidneys and the explosiveness of outbreak of epidemic mortality. The zero order correlation here is $r_{13c} = +0.447 \pm 0.093$. The difference between the zero order and the sixth order coefficients is 0.140.

4. Turning to diseases of wholly different etiology from those dealt with up to this point, namely, typhoid fever and cancer, the correlations between the normal death rate from these diseases and the explosiveness of outbreak of epidemic mortality are found to be of an entirely different order of magnitude. In neither case, typhoid or cancer, is the coefficient sensibly different from zero, having regard to its probable error. Clearly, whatever factors, innate or environmental, or both, are measured by these death rates can not have had any sensible influence in determining the suddenness with which the mortality curve rose during the influenza epidemic. It is generally held on good grounds that the typhoid death rate is an excellent index of the general sanitary status of a community. If it may be so accepted in the present connection, the result just stated bears out in precise mathematical terms what was obvious to the thoughtful and candid observer at the time of the epidemic, namely, that the severity with which a city was hit by the epidemic bore no relation to its general sanitary status or to the efficiency of its health organization. In this connection there is a further interesting mathematical point regarding the typhoid correlation. The zero order gross correlation for this death rate with I_6 is $r_{13d} = + 0.342 \pm 0.102$. This is a probably statistically significant correlation; but observe that as we make the cities constant in respect to demographic and environmental factors the reduction in the correlation is very great, ending with the sixth order coefficient at a value $r_{13d \cdot 456789} = + 0.105 \pm 0.114$, a drop of 0.237 in the coefficient. But this is exactly what would be expected when it is recalled to what an extent the typhoid death rate of a community depends upon the environmental conditions in that community.

5. The normal cancer death rate is not significantly correlated with the epidemicity at any stage, nor is the correlation altered to any extent by making the demographic and environmental factors constant. This is indicated by the fact that the zero order coefficient is $r_{13e} = + 0.235 \pm 0.109$, while the sixth order coefficient is lower by only 0.094.

6. Taking all the results together, it is seen that a considerable part of the high correlation found for the tuberculosis death rate with epidemicity index disappears if the cities are made constant and equal in respect to some six important demographic and environmental factors. The correlation is still, however, in spite of the reduction, significant in comparison with its probable error. Death rate from all causes is highly correlated under all circumstances with explosiveness of outbreak. This death rate may be taken as an index of the general healthfulness of the community.[12] But the out-

[12] It is so used, for example, by Brownlee, J., An investigation into the epidemiology of phthisis in Great Britain and Ireland, *Med. Res. Comm. Spec. Rept.*, Ser. no. 46, pp. 1–98, 1920.

standing *single* factor, which apparently more than any other one thing yet discovered, determined how abruptly or explosively the mortality was to rise at the outbreak of the epidemic, was the normal death rate in the community from organic diseases of the heart.

DESTRUCTIVENESS OF THE 1918 EPIDEMIC

As explosiveness of outbreak of epidemic mortality is obviously a characteristic which might conceivably be nearly or quite distinct and independent from destructiveness as measured by the total number of persons killed by the epidemic, it is a matter of doubt what may be the real meaning of the biological conclusions flowing from the peculiar facts just brought out. The obvious answer to this point is to study destructiveness in the same way that we have explosiveness of outbreak, by the method of multiple correlation.

The problem may then be stated in the following terms: The 34 large American cities for which we have available published weekly data, varied enormously among themselves in respect to the destructiveness of the epidemic, as measured by the number of their inhabitants who died in excess of the normal, during the autumn and winter of 1918–1919. What factor significantly influenced or determined this variation?

The methods by which this problem may be attacked are the same as those already used in the preceding section.

Variables discussed

The variables discussed are precisely the same as those listed on page 474, with two exceptions. These are (*a*) that here the variable I_6 (explosiveness of outbreak) is omitted, and (*b*) its place is taken by a new variable, destructiveness, which is indicated by the subscript 2 in all that follows. Destructiveness is measured by the twenty-five week excess mortality rates calculated and published by the Bureau of the Census.[13] These twenty-five-week excess rates indicate the number of people dying from all causes, during the twenty-five weeks following the initial outbreak of the epidemic in this country in the autumn of 1918, in excess of the number who probably would have died in the same period had no epidemic occurred. The rates for the 34 cities are given in table 138, and hence need not be reprinted here. The values of the other variables are given in table 152.

[13] *Cf. Pub. Health Repts.*, vol. 34, no. 11, p. 505, 1919.

Demographic and environmental correlations

Just as in the discussion of explosiveness of outbreak, we may consider first the net fifth order correlations of destructiveness with the several demographic and environmental factors for which we have data. The coefficients are exhibited in table 155.

The results here are clear cut. The only variable having a net coefficient which can be regarded as sensibly different from zero is latitude. There the coefficient is certainly statistically significant. Taken at its face value, and remembering the significance of the negative sign, this means that the farther south the city the greater the rate of mortality in excess of the normal. Or put in another way, the coefficient indicates that the influenza epidemic had a definite, though at best not marked, tendency to be more destructive of life in southern than in northern latitudes. Too much stress

TABLE 155

Net correlation of destructiveness (25-week excess mortality) with various demographic and environmental factors

VARIABLE CORRELATED WITH DESTRUCTIVENESS (25-WEEK EXCESS MORTALITY)	r SUBSCRIPTS	COEFFICIENT
Age distribution of population	24.56789	$+0.132\pm0.114$
Sex ratio of population	25.46789	$+0.161\pm0.113$
Density of population	26.45789	$+0.163\pm0.113$
Latitude of city	27.45689	-0.424 ± 0.095
Longitude of city	28.45679	-0.133 ± 0.114
Rate of growth of population, 1900–1910	29.45678	-0.083 ± 0.115

must not be laid upon this result, however, because of the peculiar spatial distribution of this group of 34 cities. This point has been fully discussed already and need not be dwelt on again here. The same reasons exist for suspending judgment as to the full biological significance of the apparently definite correlation between destructiveness of the epidemic and latitude, as were emphasized in the case of the similar correlation between explosiveness of outbreak and latitude.

The remaining coefficients are very definitely so small as to leave no doubt about their meaning. Throughout the long process of making more and more variables constant and getting the successive higher order net coefficients, no one of these demographic characters, except latitude, ever showed a value sensibly differing from zero. This fact is well indicated by the zero order coefficients, which are as follows:

$r_{24} = +0.024 \pm 0.116$ (age)
$r_{25} = -0.029 \pm 0.116$ (sex)
$r_{26} = +0.111 \pm 0.114$ (density)
$r_{27} = -0.325 \pm 0.103$ (latitude)
$r_{28} = +0.0007 \pm 0.116$ (longitude)
$r_{29} = -0.071 \pm 0.115$ (growth)

It can, then, be safely asserted that in the determination of the variation among these 34 large American cities in respect to the excess mortality due to the epidemic, the age and sex distribution of the population, its degree of crowding, its rate of recent growth, or the distance of the city west from the Atlantic seaboard, played no appreciable part whatever. This conclusion is true whether all these factors were allowed to vary together, or whether, as in a laboratory experiment, one to five of them were held constant, while the net influence of one or more varying alone was tested.

Whether or not the same conclusion will hold generally for other cities of a different order of size, or for rural districts, remains to be shown by further work. But as to the facts for the 34 cities listed in table 152, there can be no doubt or argument.

TABLE 156

Net correlation of destructiveness (25-week excess mortality) with the normal death rate from certain specified causes.

VARIABLE CORRELATED WITH DESTRUCTIVENESS (25-WEEK EXCESS MORTALITY); DEATH RATE FROM:	r SUBSCRIPTS	COEFFICIENT
All causes	23f. 456789	+0.405±0.097
Pulmonary tuberculosis	23a. 456789	+0.279±0.107
Organic diseases of the heart	23b. 456789	+0.537±0.082
Nephritis and Bright's disease	23c. 456789	−0.008±0.116
Typhoid fever	23d. 456789	−0.138±0.113
Cancer and other malignant tumors	23e. 456789	+0.268±0.107

Death rate correlations

Turning to the same set of normal death rate variables as were studied in connection with explosiveness we have the results set forth in table 156.

From this table we note the following points:

1. Epidemic excess mortality is significantly correlated with the normal death rate from all causes in these cities. This is true in gross ($r_{23f} = +0.435 \pm 0.094$) and also when the environmental and demographic factors listed in table 155 above are held constant, as in an experiment. In other words, the number of people dying during the epidemic in each of these 34 cities was determined to a significant, though not a high, degree by the

usual mortality relations of the community, as indicated by the normal death rate from all causes. Cities which have normally a high death rate had also a relatively high mortality from the influenza epidemic, and *vice versa* those normally enjoying a relatively low mortality rate lost but relatively few persons in the epidemic. It is to be noted, however, that while the net coefficient $r_{23f.456789}$ has a value more than 4 times its probable error and therefore is to be regarded as certainly significant statistically, yet this correlation is lower than the corresponding one for explosiveness of outbreak and death rate from all causes (*cf.* table 154). We have:

Explosiveness, $r_{13f.456789}$ =	$+0.572 \pm 0.078$
Destructiveness, $r_{23f.456789}$ =	$+0.405 \pm 0.097$
Difference	0.167 ± 0.124

While this difference is not significant in comparison with its probable error, nevertheless there is considerable probability that with a larger sample it would become so. It thus appears that the explosiveness of outbreak of the epidemic mortality was, perhaps, somewhat more influenced by the normal mortality rate of the community than was its total magnitude or destructiveness.

2. The total destructiveness of the epidemic is not significantly correlated with the normal death rate of the community from pulmonary tuberculosis when the important demographic and environmental factors listed in table 155 are held constant. Here we come upon a distinct break between the two epidemiological characteristics, explosiveness and total destructiveness. The former is, and the latter is not, significantly correlated with the normal tuberculosis death rate. A difference of the same sense is evident in the zero order gross correlations, which are $r_{13a} = + 0.578 \pm 0.077$ and $r_{23a} = + 0.428 \pm 0.094$. Both of these gross values are more than 3 times the probable error, but owe a considerable portion of their high values, as is demonstrated by the sixth order coefficients, to intercorrelations with other variables.

3. The highest net correlation of destructiveness of the epidemic is with the normal death rate from organic diseases of the heart. When all of the demographic and environmental factors with which we are dealing are held constant for these cities, we find a correlation of $+ 0.537 \pm 0.082$, a value nearly 7 times its probable error, between these two variables. The coefficient is higher than that for the normal death rate from all causes with destructiveness. It very clearly appears that of the 12 different factors here studied the normal death rate of the community from organic diseases of the heart had more to do with determining the proportionate part of the

population which perished in the epidemic than any other factor. In those cities having normally a high heart-disease rate a relatively large number died in the influenza epidemic, and *vice versa*. The same thing was shown to be true for explosiveness of outbreak of epidemic mortality. The condition of the population in respect to cardiac soundness played a significant rôle in determining the suddenness and frequency with which people died during the autumn and winter of 1918, when the 34 large American cities were struck by the influenza epidemic. It is interesting to note that the net sixth order correlation coefficient is higher in the case of organic diseases of the heart than the gross zero order coefficient, which is $r_{23b} = +0.487 \pm 0.088$. This means that in the gross, or zero order coefficient, the true organic relationship existing between destructiveness of epidemic and normal cardiac death rate is obscured by the fact that there is a high correlation between the latter variable and the age distribution of the population ($r_{3b4} = + 0.609 \pm 0.073$), the meaning of this coefficient being, of course, that the higher the average age of a population, the higher the death rate from organic diseases of the heart, and *vice versa*—a relationship which would be expected *a priori* from what we know about cardiac affections. As soon as we make the cities all constant in respect to age distribution of the population, we get a marked increase in the correlation coefficient between epidemic destructiveness and normal cardiac rate ($r_{23b\cdot4} = + 0.596 \pm 0.075$). This is an increase of 0.109 in the coefficient. But, as there is also a sensible negative correlation in this group of 34 cities between normal death rate from organic heart diseases and latitude ($r_{3b\cdot7} = - 0.300 \pm 0.105$), the high value of $r_{23b\cdot4} = + 0.596 \pm 0.075$ is reduced somewhat when the cities are made constant in respect to both age distribution and latitude, the coefficient being $r_{23b\cdot47} = + 0.549 \pm 0.081$. The other demographic and environmental variables have only negligible effect upon the r_{23b} correlation.

4. The net correlation between destructiveness of the epidemic and the normal death rate from diseases of the kidneys is sensibly zero. Here again there is a marked contrast between explosiveness of epidemic mortality and destructiveness (*cf.* table 154). The gross zero order correlation between destructiveness and normal death rate from kidney diseases ($r_{23c} = + 0.282 \pm 0.106$) is less than three times its probable error, as well as the net sixth order coefficient.

5. The destructiveness of the epidemic is not significantly correlated with the normal death rate from either typhoid fever or cancer, either net, when all the demographic and environmental variables are held constant, or in gross ($r_{23d} = + 0.014 \pm 0.116$, and $r_{23e} = + 0.215 \pm 0.110$). We

observe the same contrast in the correlations of these diseases with destructiveness as we did when they were correlated with explosiveness, in comparison with the correlation of normal death rate from organic diseases of the heart with these same epidemiological characteristics.

THE CORRELATION BETWEEN EXPLOSIVENESS AND TOTAL DESTRUCTIVENESS OF THE EPIDEMIC MORTALITY.

The results already set forth indicate clearly that explosiveness of outbreak of epidemic mortality (as measured by the epidemicity index I_6) and total destructiveness of the epidemic (as measured by the twenty-five-week excess mortality) are distinct epidemiological characters. The question then presents itself as to how closely these two characters are correlated. To the answer of this question we may now turn.

For the 34 large American cities furnishing the material set forth in table 152 we have data on the following variables:

SUBSCRIPT NUMBER	VARIABLE
1	Explosiveness of outbreak of epidemic mortality, I_6
2	Destructiveness (twenty-five-week excess mortality)
3a	Normal death rate from pulmonary tuberculosis
3b	Normal death rate from organic heart diseases
3c	Normal death rate from acute nephritis and Bright's disease
3d	Normal death rate from typhoid fever
3e	Normal death rate from cancer and other malignant tumors
3f	Normal death rate from all causes
4	Age distribution of population
5	Sex ratio of population
6	Density of population
7	Latitude
8	Longitude
9	Rate of growth of population, 1900–1910

The gross or zero order correlation between explosiveness of epidemic mortality (I_6) and total destructiveness (25-week excess mortality) is

$$r_{12} = +0.709 \pm 0.058$$

This obviously represents a relatively high, but by no means perfect, correlation. In general, it means that cities having an incidence of epidemic mortality more sudden and explosive in its outbreak than the average were highly likely to have a total mortality from the epidemic above the average, and *vice versa.*

It is essential, however, just as before, to find the *net* value of this correlation when the various environmental, demographic, and biological variables listed are held constant as in an experiment. The initial step in such a process is to calculate the first-order coefficients, where successively, one at a time, each variable in the series is held constant while the correlation between variables 1 and 2, in which we are interested, is determined. In table 157 are given these first-order correlation coefficients between the variables 1 and 2 (explosiveness and destructiveness).

It is evident from this table that making any single one of the 12 variables constant has little effect upon the correlation between explosiveness and destructiveness of the epidemic mortality. The coefficients are all relatively high.

TABLE 157

First-order correlations between explosiveness (I_6) and destructiveness (25-week excess mortality) of the 1918 influenza epidemic in 34 American cities

r SUBSCRIPTS	COEFFICIENT
12.3a	$+0.626 \pm 0.070$
12.3b	$+0.592 \pm 0.075$
12.3c	$+0.679 \pm 0.062$
12.3d	$+0.750 \pm 0.051$
12.3e	$+0.694 \pm 0.060$
12.3f	$+0.626 \pm 0.070$
12.4	$+0.718 \pm 0.056$
12.5	$+0.736 \pm 0.053$
12.6	$+0.707 \pm 0.058$
12.7	$+0.687 \pm 0.061$
12.8	$+0.729 \pm 0.054$
12.9	$+0.723 \pm 0.055$

Let us examine the effect of making *all* the demographic and environmental factors (subscripts 4 to 9, inclusive) constant at the same time. The coefficient is

$$r_{12 \cdot 456789} = +0.706 \pm 0.058$$

This is almost absolutely identical with the zero order gross coefficient given above. This result means that the factors age and sex distribution, density and rate of recent growth of the population, latitude and longitude of the city (with all implied climatic differences), have no influence in determining the correlation between explosiveness and total excess mortality rate.

Taking in the biological (normal death rate) variables we have the following seventh-order coefficients:

$$r_{12\cdot 3a456789} = +0.675 \pm 0.063$$
$$r_{12\cdot 3b456789} = +0.579 \pm 0.077$$
$$r_{12\cdot 3c456789} = +0.711 \pm 0.057$$
$$r_{12\cdot 3d456789} = +0.749 \pm 0.051$$
$$r_{12\cdot 3e456789} = +0.700 \pm 0.059$$
$$r_{12\cdot 3f456789} = +0.632 \pm 0.069$$

It is obvious that these normal death rates influence very little, either one way or the other, the correlation between explosiveness and destructiveness of the epidemic outbreak.

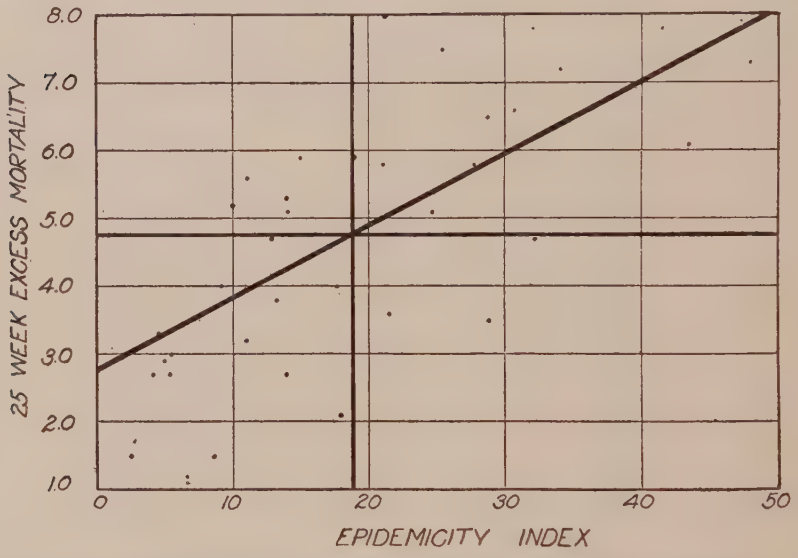

FIG. 94. REGRESSION LINE OF 25-WEEK EXCESS MORTALITY RATE OF INFLUENZA EPIDEMIC ON EXPLOSIVENESS OF OUTBREAK OF EPIDEMIC MORTALITY

One point of considerable interest which attaches to the relatively high correlation between these two variables is that one of the variables is measurable in time long before the value of the other can be possibly determined. The value of the explosiveness index I_6 can usually be determined in from two to four weeks after the beginning of the epidemic, while the total excess mortality can only be measured when the epidemic is all over. With a correlation coefficient of the magnitude of r_{12} above, one can, by means of the regression equation, make a very fair prediction of the total excess mor-

tality rate from a knowledge of the explosiveness of outbreak of the mortality measured by I_6. Actually, as the different net correlations show, we shall get practically as good a result by using the zero order coefficient and a simple equation of the form $y = a + bx$ as by employing a many-constant partial regression equation. Actually the regression equation from the zero order r_{12} is

$$D = 2.7412 + 0.1065\, I_6$$

where D denotes twenty-five-week excess mortality rate and I_6 is the epidemicity or explosiveness index. This regression line is shown graphically in figure 94.

SUMMARY

In this study there was first shown, in a variety of ways graphic and analytic, that there was an extraordinary amount of *variation* among the large cities of the country in virtually every aspect of the epidemic mortality. Some cities were much less explosively and destructively hit by the epidemic than were others. The remainder of the study was devoted to an attempt to find out some of the causes, at least, which were behind these differences.

The broad general result was first to show that such demographic and environmental factors as the age and sex constitution of the population, its density, or its recent rate of growth (used as a rough index of industrial development) had substantially nothing whatever to do with either the explosiveness or the destructiveness of the 1918 epidemic in the cities dealt with. It was, however, found that the normal death rate of each of the cities from organic diseases of the heart was correlated to a marked and significant degree with both the explosiveness of outbreak and total destructiveness of the epidemic.

Since this work was first published it has been confirmed, so far as it went, by a coöperative study under the direction of Ellsworth Huntington.[14] That study went farther and took into account the weather just preceding and during the epidemic and reaches the final conclusion that "Among all the factors yet investigated only the weather appears to have had any fundamental significance in causing the destructiveness of the epidemic to vary from city to city." While in a sort of numerical sense this conclusion appears to be warranted by the figures presented, it does not have the backing of common sense and it will, I think, be cautiously received by

[14] Huntington, E., Causes of geographical variations in the influenza epidemic of 1918 in the cities of the United States, *Bull. Nat. Res. Cncl.*, vol. 6, no. 34, pp. 1–36, 1923.

thoughtful persons. It involves as one of its major supporting elements in the statistical analysis, a rather high correlation between the *normal*, regular death rate of the community from organic heart disease (and also for that matter the normal tuberculosis death rate) and the state of the weather for ten weeks in 1918 at the time of the epidemic! Now just *possibly* the state of the weather determines the death rate from organic heart disease at a particular time. But surely it is difficult to suppose that ten weeks of weather in the autumn of 1918, however bad it may have been, can possibly have influenced the organic heart death rate of 1915, 1916, and 1917! And equally it is difficult to suppose that both of these phenomena (*normal* organic heart death rate and weather for 10 weeks in 1918) can depend upon a common cause. Yet something like one or the other of these equally difficult statements will have to be accepted by anyone who accepts Dr. Huntington's final conclusion.

CHAPTER XVIII

The Incidence of Influenza among the Tuberculous[1]

A problem of great interest in connection with the influenza pandemic of 1918 may be stated in this way: Were actively tuberculous persons more or less likely to contract influenza during the epidemic than non-tuberculous members of the general population living under essentially the same environmental conditions, and equally exposed to infection? The question may be argued *a priori* either way, and in the period since the epidemic a good deal of what can in fairness only be called wholly unscientific and inconclusive has been written about it. It can only be answered by a sufficiently large collection of critically adequate statistics.

The purpose of this chapter is to present certain *ad hoc* statistical material collected in this laboratory.

The data were collected by the Visiting Tuberculosis Nurses of the Baltimore Health Department, working on this problem under the direction and supervision of Miss Blanche F. Pooler, supervisor of field workers in the writer's laboratory. Each household containing an active case of tuberculosis registered with the Health Department was visited by a nurse and a card form filled out by her, after personal determination of the facts about the family.

Each card brought in by a nurse was critically scrutinized by Miss Pooler, and if any information was lacking, or if the facts recorded were in any respect unusual, the nurse was sent back on a second or even a third visit, until finally the data accepted as of final record were as nearly accurate as it was humanly possible to make them.

Tables 158 to 160 inclusive comprise the preliminary tabulations which it is desired to present here. Only the figures for whites are given in this note. The numbers in the tables for negroes were too small to warrant taking space for a brief preliminary communication. The negro data show

[1] This chapter is a reprint of a paper with the title, "Preliminary note on the incidence of epidemic influenza among the actively tuberculous," published in *Quart. Publ. Amer. Stat. Assoc.*, vol. 16, pp. 536–540, 1919. When it was published it was expected that it would be followed by a much more complete presentation and analysis of the statistical material that had been collected. Unfortunately before this could be done the original card records were destroyed by fire. The material must therefore remain in the incomplete form here presented.

no essential differences from the whites in respect to the main points here under discussion. The tables are arranged on the principle of dichotomy to get a maximum of information in a minimum of space. A description of the second line of table 158 will indicate how this table is to be read. In households of two persons each there were 14 tuberculous persons who had influenza and 120 tuberculous persons who did not have influenza. Of the

TABLE 158

Showing the incidence of influenza among tuberculous and non-tuberculous white individuals, arranged (a) by number of persons in household, and (b) by presence or absence of other cases of influenza

NUMBER IN HOUSEHOLD	TUBERCULOUS				NOT TUBERCULOUS			
	Influenza		No influenza		Influenza		No influenza	
	Other cases in household	No other cases in household	Other cases in household	No other cases in household	Other cases in household	No other cases in household	Other cases in household	No other cases in household
1				14				
2	4	10	12	108	4	15	7	100
3	46	39	38	161	76	22	118	292
4	72	28	81	255	168	37	243	696
5	89	27	78	221	262	21	363	749
6	73	16	96	210	303	29	419	822
7	71	9	83	123	358	18	480	636
8	51	2	68	82	257	24	414	446
9	22	3	40	33	117	8	188	246
10	18	1	16	20	114	3	138	170
11	8		12	12	49	5	91	43
12	3		5	5	36	1	63	43
13	2		3	2	32		28	24
14								
15	1		1	1	12		16	14
Total...	460	135	533	1,247	1,788	183	2,568	4,281
	595		1,780		1,971		6,849	
	2,375				8,820			

14 tuberculous persons who had influenza 4 were in households where other cases of influenza occurred, and 10 were in households where no other cases of influenza except their own occurred. Again, in households of two persons, there were 19 non-tuberculous persons who had influenza and 107 who did not. Four of the influenza cases occurred in households where there were other cases, and 15 of the cases were in households where there were no others, etc.

From table 158 we note that of the 2375 tuberculous persons, 595, or 25 per cent, had influenza, while 1780, or 75 per cent, did not have the disease during the epidemic. Of the 8820 non-tuberculous individuals living in the same households as the tuberculous, 1971, or 22.3 per cent, had influenza,

TABLE 159

Showing the incidence of influenza among white tuberculous persons according to (a) duration of the tubercular process, and (b) presence or absence of other cases of influenza in the household

YEARS PATIENT HAD BEEN TUBERCULOUS	PATIENT HAD INFLUENZA			PATIENT DID NOT HAVE INFLUENZA		
	Other cases in household	No other cases in household	Total	Other cases in household	No other cases in household	Total
Under 1 year....	108	65	173	57	133	190
1 year.....	44	17	61	68	172	240
2 years.....	65	15	80	75	176	251
3 years.....	63	20	83	64	139	203
4 years.....	50	15	65	65	128	193
5 years.....	56	11	67	46	135	181
6 years.....	39	14	53	49	109	158
7 years.....	40	10	50	36	76	112
8 years.....	40	11	51	37	74	111
9 years.....	35	10	45	45	110	155
10 years.....	13	4	17	11	31	42
11 years.....	1	2	3	7	17	24
12 years.....	6		6	8	16	24
13 years.....	1		1	2	2	4
14 years.....	2		2		3	3
15 years.....				3	7	10
16 years.....	1		1	3		3
17 years.....					1	1
18 years.....	1		1			
20 years.....				2		2
23 years.....					1	1
25 years.....					1	1
32 years.....					1	1
34 years.....	1		1			
Total.....	566	194	760	578	1,332	1,910

and 6849, or 77.7 per cent, did not have it. It, therefore, appears that, under the same environmental conditions of living, only 2.7 per cent more of the tuberculous individuals than of the non-tuberculous contracted influenza during the epidemic. This difference is small and probably not statistically significant.

Table 159 concerns the tuberculous *only*. It arranges the data to show the relative liability of a tuberculous person to contract epidemic influenza, in relation to the length of time he (or she) had been tuberculous.

This table shows among other things that the longer had been the duration of the tubercular process the smaller in general was the probability that the patient would have influenza during the epidemic, though in durations of one to nine years inclusive the proportionate incidence of influenza changed but slightly.

From table 160 we note:

TABLE 160

Showing the incidence of influenza among tuberculous and non-tuberculous whites according to the number of cases of influenza in the household

NUMBER OF CASES OF INFLUENZA IN HOUSEHOLD	NUMBER OF HOUSE-HOLDS	TOTAL NUMBER OF PERSONS IN HOUSE-HOLD	TOTAL CASES OF INFLU-ENZA	PERSONS TUBERCULOUS			PERSONS NOT TUBERCULOUS		
				Had influenza	Did not have influenza	Total	Had influenza	Did not have influenza	Total
0	1,104	5,492	0	0	1,234	1,234	0	4,258	4,258
1	324	1,644	324	137	236	373	187	1,084	1,271
2	228	1,231	456	132	141	273	324	634	958
3	152	844	456	113	71	184	343	317	660
4	88	546	352	65	35	100	287	159	446
5	73	536	365	68	27	95	297	144	441
6	46	358	276	40	13	53	236	69	305
7	26	218	182	33	5	38	149	31	180
8	14	131	112	14	3	17	98	16	114
9	4	42	36	3	2	5	33	4	37
10									
11									
12									
13	1	13	13	1		1	12		12
Total....	2,060	11,055	2,572	606	1,767	2,373	1,966	6,716	8,682

1. That a little more than one-half (1104) of the total number of *households* (2060) comprised in the statistics had no case of influenza in them during the epidemic.

2. That only about one-fourth (2572) of the total *persons* (11,055) included in the statistics had influenza.

3. That in households where only one case of influenza occurred, 37 per cent of the tuberculous persons living in such households had influenza, while but 15 per cent of the non-tuberculous living in such households had influenza.

4. That in households where two cases of influenza occurred, 48 per cent of the tuberculous persons, and 34 per cent of the non-tuberculous living in such households had influenza.

5. That in households where three or more cases of influenza occurred, 68 per cent of the tuberculous persons and 66 per cent of the non-tuberculous living in such households contracted influenza.

CHAPTER XIX

Epidemic Encephalitis[1]

During the last years of the war and since there has occurred the significant beginning and menacingly rapid spread of a disease either previously not existing, or at least not occurring frequently enough to get general recognition, namely, *encephalitis lethargica* or epidemic encephalitis. Because of its extremely rapid development in frequency of occurrence and because of its extremely fatal character, together with the probability that it may become an epidemic disease of an importance similar to that of poliomyelitis it seems desirable, thus early in its history, to make some analysis of its statistical characteristics.

The aid which the mathematical analysis of statistical data can render the epidemiologist and pathologist, and the degree of certainty which such analysis can add to their conclusions, are not generally recognized. Usually the health officer is content with the mere tabulation of frequency of occurrence of morbidity and mortality. Such figures are too frequently made the basis of conclusions without any recognition of the errors of sampling involved. Before one can be in any degree certain of conclusions from such figures their probable errors must be known.

MATERIAL

Two papers have appeared giving raw data regarding the development of epidemic encephalitis. The first of these papers[2] deals with the cases of the disease in the whole United States in the years 1918 and 1919. The second paper[3] deals with the much more restricted field of New York City in 1919 and 1920, but because of the accelerated frequency of occurrence of the disease during the latter year, contains far more statistical material than Smith's paper. It is with the data from this second paper that we shall chiefly deal here.

[1] The paper on which this chapter is based originally appeared under the title, "A statistical note on epidemic encephalitis," in *Johns Hopkins Hosp. Bull.*, vol. 32, pp. 221–225, 1921. It is reprinted here practically without change.

[2] Smith, H. F., Epidemic encephalitis (encephalitis lethargica, nona), Report of studies conducted in the United States, *Pub. Health Repts.*, vol. 36, pp. 207–242, 1921.

[3] Epidemic encephalitis in New York City. *Weekly Bulletin Department of Health, New York City*, N.S., vol. 10, no. 12, pp. 89–92, March 19, 1921.

CASE FATALITY RATE

Table 161 gives the incidence and deaths from this disease in New York City in 1919 and 1920. There is a slight correction of an obvious arithmetic error in the total deaths in 1919, from that given in the original.

These data give the following case fatality rates:

$$1919, \text{ case fatality rate} = \frac{33 \times 100}{128} = 26 \text{ per cent}$$

$$1920, \text{ case fatality rate} = \frac{211 \times 100}{565} = 37 \text{ per cent}$$

TABLE 161

Incidence and deaths from epidemic encephalitis, by months, 1919–1920

MONTH	1919		1920	
	Cases	Deaths	Cases	Deaths
January	5		36	12
February	16		149	50
March	25	9	116	52
April	10	1	66	16
May	16	5	42	22
June	4	1	20	12
July	2	1	39	9
August	1		28	11
September	8	5	22	6
October	23	7	11	3
November	9	3	12	9
December	9	1	24	9
Totals	128	33	565	211

The first of these figures agrees well enough with the 29 per cent given by Smith for the case fatality rate over the whole country in the years 1918 and 1919. The significant thing is that the case fatality rate appears to be increasing. Such a conclusion depends upon the assumption that morbidity from this cause was at least as well reported in 1920 as in 1919. It may be accepted with reasonable certainty that the *deaths* were as well reported in the one year as the other. The well-known high standards maintained in the work of the Bureau of Vital Statistics of the New York City Department of Health practically ensures this. The morbidity figures are more doubtful, because this disease has only recently been made reportable in the city. But let us carefully examine the alternatives which are presented. If we assume (*a*) that the deaths have been equally well reported in 1919 and

1920, which is a justifiable assumption, and (*b*) that the true, but unknown, case-fatality rate has remained absolutely constant at the 1919 figure during these two years, then it follows that to get the 37 per cent observed, it will be necessary to suppose that the 565 cases reported in 1920 far *underestimate* the true number which occurred. It must be assumed that actually 812 cases occurred instead of the 565 reported. Now it would seem altogether unlikely, indeed indefinitely improbable, that in the year 1920 there occurred in New York City 247 cases of encephalitis lethargica about which the Department of Health did not know. The *prima facie* reason why this is so improbable is the ever-increasing interest and the recognition of the importance of this disease by the Department of Health during the period named.

Suppose we consider the second alternative which is that the figures for 1919 greatly underestimate the true and assumed constant case fatality rate. This means, if we assume the deaths to have been substantially accurately reported for the same reason as before, that there were more *cases* reported in 1919 than actually occurred. To justify this second alternative it must be supposed that actually there occurred in 1919 in New York City only 89 cases of epidemic encephalitis, instead of the 128 reported. This would be a highly improbable assumption. And further we must reckon with the close agreement between Smith's figures of 29 per cent for the case fatality rate for the whole country and the New York 1919 figures of 26 per cent.

Altogether it may be tentatively concluded, with a high degree of probability, that the case fatality rate from the disease is on the increase. But the New York numbers are absolutely small in a statistical sense, and the further question must be asked, as to whether the 11 per cent increase in the case fatality rate between 1919 and 1920 in New York is statistically significant, *i.e.*, greater than might reasonably be supposed to have arisen from chance fluctuation alone.

The problem may be stated in this way: If in a sample of 128 cases of the disease 33 deaths occur, what would be the number of deaths expected in a sample of 565 cases, if nothing but chance were involved in the matter, or in other words if there had been no change in the true case-fatality rate?

We have, by well understood principles:[4]

$$\text{Mean deaths expected in second sample of } 565 = 148 \pm 16$$

[4] Pearson, K., On the influence of past experience on future expectation. *Phil. Mag.*, May, 1907, pp. 365–378.

Pearl, R., A statistical discussion of the relative efficacy of different methods of treating pneumonia, *Arch. Int. Med.*, vol. 24, pp. 398–403, 1919.

Or, in other words, it is an even chance, if chance alone caused in 1920 fluctuations from the case-fatality rate observed in 1919, that from 132 to 164 deaths would have occurred in 1920, rather than a number smaller than 132 or larger than 164. Actually 211 deaths occurred in 1920. The difference between 211 and 148 is more than 3.9 times the probable error of the latter figure. Whence we may conclude that there was a statistically significant rise in the case fatality rate in 1920 as compared with 1919 in New York City.

SEASONAL DISTRIBUTION

A question which at once occurs to one in examining table 161 is this: Is the seasonal distribution (by months) of cases changing as the disease grows in importance, or is the monthly distribution in 1920 different from that of 1919 only by as much as might reasonably be expected from chance sampling?

To determine this question it is necessary only to determine the probability that the two distributions differ significantly from each other. It has been shown by Pearson[5] that such a probability will be given by the expression

$$P = \frac{\int_{\chi}^{\infty} \chi^{q-2} e^{-\frac{1}{2}\chi^2} d\chi}{\int_{0}^{+\infty} \chi^{q-2} e^{-\frac{1}{2}\chi^2} d\chi}$$

where

$$\chi^2 = NN'S\left[\frac{(f/N - f'/N')^2}{f+f'}\right]$$

the summation S extending over all pairs of the elemental frequencies f and f'.

[5] Pearson, K., On the criterion that a given system of deviation from the probable in the case of a correlated system of variables is such that it can be reasonably supposed to have arisen from random sampling, *Phil. Mag.*, pp. 157–175, 1900.

Pearson, K., On the probability that two independent distributions of frequency are really samples from the same population, *Biometrika*, vol. 8, pp. 250–254, 1911.

Pearson, K, On a brief proof of the fundamental formula for testing the goodness of fit of frequency distributions, and on the probable error of "P", *Phil. Mag.*, vol. 31, pp. 369–378, 1916.

From the data of table 161, we get for the probability that the 1920 and 1919 distribution are two random samples from the same unchanged phenomenal universe

$$\chi^2 = 89.542$$

whence it is easily determined that

$$P < 0.000,000,1$$

Or, in words, it appears that less often than once in 10 million trials would one expect to get true samples of the size here dealt with, as divergent as are

TABLE 162

Encephalitis cases and deaths, by age and sex, 1920

AGES	CASES			DEATHS		
	Male	Female	Total	Male	Female	Total
Under 5 years	23	24	47	10	8	18
5–9 years	26	14	40	10	2	12
10–14 years	32	18	50	6	5	11
15–19 years	23	24	47	5	5	10
20–24 years	37	34	71	13	11	24
25–29 years	29	27	56	11	14	25
30–34 years	35	18	53	8	8	16
35–39 years	30	18	48	14	11	25
40–44 years	19	14	33	7	10	17
45–49 years	24	16	40	13	7	20
50–54 years	15	8	23	8	3	11
55–59 years	8	3	11	6	1	7
60–64 years	7	7	14	3	5	8
65–69 years	4	4	8	2	1	3
70–74 years	3	2	5	2	1	3
75–79 years	0	1	1	0	1	1
80–84 years	1	1	2	0	0	0
85 years and over						
Totals	316	233	549	118	93	211

the 1919 and 1920 monthly distributions, from the operation of chance alone. Hence we conclude that there was a significant difference in the seasonal incidence of the disease, as indicated by the monthly distribution of cases, in 1919 as compared with 1920 in New York. Examining the details of the χ^2 calculations it is seen that the significant divergences are in the autumn and spring. There was a significant flare-up in the spring of 1920 lacking in 1919, and a significant autumn outbreak in 1919 lacking in 1920.

TIME LAG OF DEATHS

On the basis of the twenty-four months' experience exhibited in table 161 it is possible to determine the average time that deaths lag behind cases. Put in another way this means the mean duration of *fatal cases* of encephalitis lethargica, between recognized onset and death. This is found to be:

Mean duration of time between onset and death $= 1.046 \pm 0.230$ months

The epidemiological significance of this datum is that it may be expected, in so far as the 1919 and 1920 experience of New York City may be regarded as typical, that the peak of mortality in an epidemic outbreak of this disease will probably occur from twenty-three to thirty-seven days after the peak of morbidity has been passed. The contrast of this with the time lag in epidemic influenza is sufficiently striking.

INFLUENCE OF SEX

Table 162, gives the sex and age distribution of the cases and deaths in New York City in 1920.

From these data the first question to which attention may be directed is this: Is the difference between the sexes in respect of (*a*) incidence, or (*b*) fatality, greater than might reasonably be expected to arise from chance alone, in a city having the sex distribution of its population that New York has?

By elementary principles of probability it is easily found that, among those persons having the disease in New York in 1920, the probability that a given person picked at random would be a male is given by

$$p = \frac{316}{549} = 0.5756$$

whence

$$\frac{pq}{n} = 0.000445$$

In the whole population of New York (on the basis of the 1910 census figures) the probability that any person picked at random will be a male is

$$p' = 0.4994$$

and

$$\frac{p'q'}{n'} = 0.000,000,001$$

whence

$$p - p' = 0.0762 \pm 0.0142$$

and

$$\text{Diff./P.E. Diff.} = 5.4$$

Or, it appears that there is a significantly larger proportion of males among those attacked by epidemic encephalitis than there is among the general population. A deviation as large as that between p and p' would occur on the basis of chance alone only once in about every 10,000 trials.

Among those *dying* from epidemic encephalitis in New York City in 1920 the probability that any individual picked at random would be a male was

$$p = \frac{118}{211} = 0.5592$$

Whence

$$\frac{pq}{n} = 0.001168$$

$$p' = 0.4994 \text{ as before}$$

$$p - p' = 0.0598 \pm 0.0230$$

From this result we conclude that the proportion of males dying from epidemic encephalitis in New York in 1920 was not significantly different from that found in the general population.

It thus appears that more males were attacked but no more died from encephalitis than would have happened if sex had no differential effect whatever relative to this disease.

INFLUENCE OF AGE

It is stated that "no age seems exempt, but the greatest proportion of cases occurred in young adults, with a preponderance of males." This raises the following question: Is the age distribution of each of (*a*) male cases, (*b*) female cases, (*c*) male deaths, and (*d*) female deaths, significantly different, as a whole, from (*e*) the male population age distribution and (*f*) the female population age distribution?

Using the χ^2 method, and taking the 1910 age distribution of the New York City population as the basis of comparison, one gets:

For male *cases:*

$$\chi^2 = 8.254$$
$$P = 0.410$$

Or, one would expect in 4 out of every 10 trials with samples of the size here dealt with to get a divergence as great as or greater than that between the male case incidence age distribution and the general population age distribution, if epidemic encephalitis were perfectly random in its age incidence. In other words, there is slight basis for assuming that this disease attacks males more frequently at one age than another.

If we leave out the "under 5" group

$$\chi^2 = 4.111$$
$$P = 0.767$$

This means that in males five years old and over the age incidence of cases is very close indeed to a random one. In 77 out of every 100 trials there would result from chance alone deviations as great as or greater than that actually shown between male cases and the general male population.

Turning to male *deaths* we have:

$$\chi^2 = 18.450$$
$$P = 0.018$$

These values indicate a significant divergence of the male death age distribution from the general population male age distribution. But a further anlaysis gives:

For male deaths under forty-five years of age:

$$\chi^2 = 5.790$$
$$P = 0.448$$

For male deaths under thirty-five years of age:

$$\chi^2 = 3.319$$
$$P = 0.652$$

It thus appears that it is chiefly the excess of male deaths beyond age forty-five from this disease that causes the low value of P for the entire male death distribution.

Putting all the facts together it can be asserted, on the basis of the experience of New York City in 1920 with epidemic encephalitis, that in males the disease is probably not significantly more likely to attack one age than another, but instead falls upon males in about the proportion that they are normally present in the general population. It is furthermore not more fatal among the attacked at one age than another until after age 35 is passed. From that point on there are more fatal cases than would be expected if the toll of death were taken purely at random in respect of age.

Turning to the age distribution of female *cases*, and again comparing with the age distribution of the total female population (as of 1910) we get:

$$\chi^2 = 4.847$$
$$P = 0.773$$

It is thus quite certain that, so far as the experience of New York City in 1920 may be taken as typical, epidemic encephalitis occurs among females without definite age preference, striking the different ages in due proportion as they are represented in the general population. Females of any particular age are not more likely to contract the disease than females of any other age.

TABLE 163

Data for testing the deviation of the female death age distribution from the female population age distribution

AGES	m AGE DISTRIBUTION OF FEMALE POPULATION (AS OF 1910)	m' AGE DISTRIBUTION OF FEMALES DYING OF ENCEPHALITIS LETHARGICA IN 1920	$\frac{(m - m')^2}{m}$
Under 5 years	9.810	8	0.3340
5–9 years	8.553	2	5.0207
10–14 years	8.268	5	1.2917
15–19 years	9.427	5	2.0790
20–24 years	10.954	11	0.0002
25–34 years	17.495	22	1.1600
35–44 years	13.044	21	4.8526
45–64 years	12.554	16	0.9459
65 years and over	2.894	3	0.0039
Totals	92.999	93	15.6880

The age distribution of *deaths* among females presents a different case. It will be well to examine the facts in some detail. Table 163 gives the data.

$$\chi^2 = 15.6880$$
$$P = 0.048$$

It thus appears that the death distribution as a whole is significantly divergent from what would happen if the disease was as likely to be fatal at any given age as at any other. But it is also apparent that more than half of the large value of χ^2 is due to the contribution of but two age groups. These age groups are the five to nine and the thirty-five to forty-four. The deaths in the former were fewer than expected and more than expected in the latter. The numbers are small, however, and it will probably be found,

when more data are available, that the facts as to age distribution of deaths are the same in females as in males, namely that there is a definite tendency towards greater fatality in the higher age groups.

It is distinctly to be understood that the results above set forth have only such degree of validity as inheres in the experience of New York City with epidemic encephalitis during the two calendar years 1919 and 1920. Doubtless as more material accumulates new statistical relations of this disease will appear. The conclusions reached are, of course, subject to modification by more extensive experience.

What these conclusions are may be summarily stated as follows:

1. In the year 1920 the case incidence of epidemic encephalitis increased in New York City nearly five-fold over 1919. At the same time the case fatality rate increased from 26 per cent of the attacked to 37 per cent of the attacked. It is believed that this increase in case fatality rate is a real phenomenon, and it is certainly statistically significant.

2. The seasonal incidence, as judged by monthly distribution of cases was significantly different in 1920 to what it was in 1919.

3. The peak of mortality in an epidemic outbreak may be expected to occur from twenty-three to thirty-seven days *after* the peak of case incidence (morbidity).

4. There is a significantly larger proportion of males among the attacked than there is in the general population, or, put in another way, males are especially susceptible, to a statistically significant degree.

5. Deaths occur among males no more frequently in comparison with females than would be expected from the normal proportions of the two sexes in the population at large.

6. The disease is not more likely to attack either males or females at one age than at another. The age distribution of attacked cases, in other words, does not significantly differ in either sex from the age distribution of the general population.

7. The age distribution of deaths does differ significantly in both sexes from the age distribution of the population. There appears to be a definite tendency for the disease to be more fatal in the higher age groups.

CHAPTER XX

The Statistical Evaluation of Public Health Activities[1]

It must be apparent to any thoughtful student of public affairs and economics that a fundamental change is taking place in the attitude of the public mind toward altruistic social activities of all sorts. The rapid development during the last quarter century of knowledge, on the one hand, and theories on the other hand, of social betterment, in the various subdivisions of the fields of medicine, sociology, economics, etc., has had the tendency to hypnotize the public. Instinctively most right minded people like the idea of helping those who need help. They have been shown a plethora of ways in which this might be done. The cost has frequently not been counted. Indeed most of the not particularly thoughtful of our fellow citizens have tended to overlook the fact that activities intrinsically good, or supposed to be, cost anything at all. Unfortunately, however, they do. Every time a new social activity is put into operation, the public has to pay for it, either directly in the form of so-called charitable giving, or indirectly in the form of taxation.

At the present time the public is interested in and scrutinizing the cost of the altruistic activities of society as it never did before. This attitude of mind seems to me to be a healthy one, and likely to increase in extent and force. When one pays to municipality, state, and nation a distinctly noticeable fraction of his income for the privilege of being allowed to continue merely to live in that city, state, and nation, he is rightfully concerned in examining with care the question of what he gets for his money.

Just here is where arises the *necessity* of evaluating public-health activities. The person interested in and engaged in these activities cannot for a moment afford to neglect this phase of his subject, or to assume any sort of superior attitude that it is none of the public's business to inquire whether his work is worth what it costs. On the contrary he needs, if I judge the signs at all correctly, as never before to search his own business in the most critical objective way, to the end that when called up to the court of public

[1] The paper which forms this chapter first appeared under the same title in *Amer. Jour. Pub. Health*, vol. 12, pp. 915–921, 1922. It was originally delivered as an address before the Section of Vital Statistics of the American Public Health Association, at the fifty-first annual meeting, at Cleveland, Ohio, October 19, 1922.

and scientific opinion, as he will be with increasing frequency, he may be able to present his case clearly, and with intellectual cleanness.

Besides this somewhat practical reason for evaluating quantitatively the results of public-health work, there is also an intellectual and moral one, fundamentally more important. This reason may be put bluntly in this way: no such high-minded men as public-health officials are, care to engage in a dishonest business. Is our current public-health work always and everywhere honest,—I mean *really*, which is to say scientifically, honest? Do the measures we practice *really* prevent disease? Or would the course of morbific events be much the same if some of the things we do were not done? A striking illustration of my point, known to all, lies in the experience of the Providence Health Department with the fumigation of rooms with ill smelling vapors after contagious diseases. Our revered colleague, Dr. Chapin, than whom a more intellectually honest man never lived, decided to evaluate the effect of fumigation, and found it to be just exactly zero. Another example of the same point will do no harm. Dr. Levy and Dr. Freeman in Richmond could not materially reduce the summer mortality of infants in that fair city by the studious and strenuous application of all the orthodox methods of reducing infant mortality. So they instituted a campaign for the simple and homely practice of boiling diapers. It was promptly followed by a substantial drop in infant mortality. This well-known case is usually cited as an example of the intellectual genius of these workers. It seems to me to constitute an equally impressive monument to their intellectual honesty, and to furnish a splendid example of the experimental evaluation of public-health procedures, not, be it carefully noted, solely of the procedure of boiling diapers, but also, and of more consequence, of the *other* procedures.

A third reason for thought about, and frequent practice of the statistical evaluation of the results of public-health activities is that it furnishes one of the most potent means of increasing and extending knowledge that we have in this field. No one supposes that we have attained complete or sufficient knowledge of the laws of hygiene. Probably some of what is now practiced in public-health work will some time be discarded because we shall know how better to accomplish the desired result. One of the principal methods by which this result will be attained will certainly be the statistical, and chiefly because, when properly handled, it makes possible the *measurement* of the effectiveness of different procedures.

If it be granted, as I think it must, that particular attention on the part of public-health officials, to the honest, strictly scientific evaluation of the results of their activities is imperative, we may turn to the next question.

Along what lines shall this evaluation proceed? Broadly speaking, attempts in this direction have followed two lines: first, the evaluation of public-health activities in terms of mortality, and second, the evaluation in financial terms. Neither of these lines is entirely fair to the case, and so far both are unsatisfactory.

It is not fair to measure the effectiveness of public-health work entirely in terms of mortality, because much of its effectiveness in actual fact has nothing to do with mortality, but with morbidity. This fact shows itself in everyday language. We have boards of *health*, not boards of mortality, and quite rightly so. Some of the human ailments against which public-health work directs its most effective work are diseases which at the worst are not particularly fatal. An example is uncinariasis—hookworm disease. It would be folly to attempt to measure the social worth of the campaign against this distressing ailment in terms of mortality. What this work accomplishes is not primarily a reduction in mortality, but a positive increase in the sum total of human happiness and well-being, individual, social and economic. The same considerations apply to many other lines of public-health work, indeed to most of them. The most important causes of *death*, taken by and large, are not the ones against which hygiene and sanitation are, in the present state of knowledge and of the organization of society, particularly effective. But this fact should in nowise be taken to mean that public-health efforts have no great value.

The economic evaluation of public-health activities, which attempts to set up ledger accounts for each line of work, and show on one side the cost in dollars, and on the other side the supposed value of the results in dollars, seems to me again fundamentally unfair, and for essentially the same reason I have set forth above. Much, if not most, of the most valuable accomplishments of public-health work are, in the very nature of the case, not measurable or even expressible in dollars and cents. What is it worth to me in money that my children will not have diphtheria or smallpox? Who can say, or if he does attempt to say, who will attach any significance to his figures? Again, does any one find any particular real meaning in the various estimates we have had, figured out with the utmost refinements of mathematics, of the monetary loss due to tuberculosis? Lately it has been estimated[2] that "this loss means an annual charge in excess of five hundred million dollars from the curtailed longevity of individuals because of tuberculosis." This sounds interesting and impressive, because the figure is larger than any of which the human mind is capable of forming any *real* conception. But it intrigues me

[2] Dublin, L. I., and Whitney, J., On the costs of tuberculosis, *Quar. Pub. Am. Stat. Assoc.*, vol. 17, pp. 441–450, 1920.

to note that an eminent authority in quite another field estimates[3] in the same year that the annual loss to this country from the crop depredations of rats, mice, and other rodents was the same figure, $500,000,000. I do not wish in the least to criticize either estimate, both of which I am sure from some personal experience in both fields, have been made with the highest degree of accuracy attainable in our present state of knowledge, and of greater importance, attainable *in the nature of the respective cases*. But it does seem to me that most sensible persons will somehow feel that, if the true and complete losses in the two cases were really measurable, the result would *not* be to show that tuberculosis and the destruction of crops by rats are disasters of exactly the same magnitude, though this is the conclusion indicated when the economic mode of evaluation of tuberculosis is followed.

I do not mean to imply in what I have said that either the mortality or the economic modes of evaluation of public-health activities should be abandoned. Quite on the contrary both have a legitimate place and function in the matter. All I want to emphasize is that both, at the very best, tell an incomplete story, which may be quite misleading because it is incomplete. There is needed something to supplement these two. What it is, I think is apparent to all. Before we can, with anything approaching real justice and completeness, evaluate statistically the results of public-health work we must have a system of registering the statistics of *morbidity*, vastly more complete and extensive than anything we have so far had in this field. The significance of evaluations of public-health work in terms of morbidity has nowhere been more strikingly shown than in the Reports of the International Health Board. Figures 11 and 12, in their Eighth Annual Report, showing the results of anti-malaria measures in Crossett and Hamburg, Arkansas, measured in terms of number of physicians' calls for malaria, are splendid examples. My urgent plea is that this Section and this Association as a whole use every power at its command to hasten the coming of the day of adequate morbidity statistics. Splendid beginnings in this direction have been made by Dr. Dublin and Dr. Frankel working with the magnificent resources and backing of a great insurance company, and by our chairman, Mr. Sydenstricker, and his staff. But some one must make the same determined and persistent fight in this country for official morbidity statistics that Cressy L. Wilbur did for mortality statistics. Then we shall have them.

If we are agreed that we must give due attention to the statistical evaluation of our work, and that this must take account of morbidity as well as

[3] Bell, W. B., Death to the rodents, *Year Book, U. S. Dept. Agr., 1920*, pp. 421–438, 1921.

mortality and economic results, we may ask another question: What shall be our method of evaluation? Broadly, there can be only one answer to this question. We must, in the nature of the case, resort to the statistical method, either alone or combined with the experimental method. More specifically, it needs to be brought emphatically to the attention of the vital statistician and public-health officer that in the last quarter of a century the theory of statistics has advanced by leaps and bounds. The day has passed when the mere tabulation of crude statistics, and the drawing of unchecked and uncriticised conclusions from these tabulations will pass the court of scientific judgment. No statistical conclusion is today accepted by competent workers in the field, unless it is fortified by a determination of the probable errors involved. A decline in the death-rate may be absolutely large, but wholly devoid of significance, because subject to a large error of random sampling.

This is only another way of saying that until we are sure that the random fluctuations of chance might not alone have determined the value of a phenomenon which we are studying, it is idle to expect scientifically intelligent persons to believe that the value observed is due to the efforts of the health officer, to mutations in bacteria, or what not.

In the space of a fifteen-minute paper it is obviously impossible to go into the details of modern statistical technique. One can only point out that there are available now adequate text-books of statistical theory, such as those of Yule, and of Arne Fisher, and that courses in the subject are now given at our schools of hygiene and public health. One may hope that as time goes on a higher and more critical standard than now prevails will become the rule instead of the exception in the statistical operations and deductions of public-health work.

The statistical method as a means of acquiring knowledge is one of the most powerful tools at our command. At the same time it is one of the most dangerous. Every one feels entirely competent, whether he has had any special training in the recondite field of the calculus of statistics or not, to draw conclusions from figures. The result is really, though not generally recognized so to be, just as bad as would be the case if wholly untrained persons felt free to draw conclusions in the most advanced fields of physical or organic chemistry.

The statistical pitfalls most commonly overlooked in attempting to evaluate the results of public-health work are the following. More might be instanced, but these are the outstanding and crying sources of trouble.

1. It is forgotten, or deliberately disregarded, that a death-rate or a morbidity rate for a community which has not been corrected for, or as is said

technically, made specific for the age, race and sex distribution of the population of the community, may be most misleading, and lead to egregious errors.

2. Differences or trends in death-rates or morbidity rates, on the basis of either time or space, have no scientific validity or meaning till the probable errors of the differences or trends have been measured, either exactly, or at least approximately.

3. It is overlooked or forgotten that *post hoc* does not necessarily mean *propter hoc*. The human mind is prone to believe, if event B comes *after* event A, that A was the cause of B, especially when as in public-health work A was undertaken with the best of intentions, carried through with care and almost religious fervor for the purpose and in the hope that it would cause B. A great many people confuse moral convictions with physical reality. In consequence, for example, some persons are apt to get very angry if one questions in the most objective and scientific spirit, what are the causes of the decline in the tuberculosis death-rate. They take the ground apparently that because their *efforts* to reduce this mortality were sincere and honest and in the highest degree noble, therefore the decline has been in actual fact *caused* by the efforts, and that to question this purely scientific matter is to question and impugn both their motives and their efficiency. As a matter of objective scientific fact, extremely little is known about why the mortality from tuberculosis has declined.

Many examples might be given to show the sad results of confusing *post hoc* with *propter hoc* in statistical deductions in public-health work, but in the time available I can only mention two. We hear and read a great deal about the effect of "the degrading influence of poverty" upon death-rates, especially infant death-rates. Leaving aside the larger social question as to whether there is ever anything "degrading" about poverty *per se*, we may well ask about what the evidence is that poverty, in actually existing rather than hypothetical populations, increases mortality. The evidence usually given is simply that in some populations the death-rate among poor people is notably higher than among well-to-do or rich people. This is the evidence given by Hersch[4] in his recent study of the matter in Paris. But this is truly *post hoc ergo propter hoc* reasoning. It quite overlooks the fact that the rich and the poor differ statistically in any population in respect to a number of other conditions besides poverty. Until one is sure that he has adequately measured and allowed for the effect of these other differences upon the rate of mortality he cannot safely conclude that poverty is the cause of his observed differences in mortality. The weakness of the case I have cited was almost

[4] Hersch, L., L'inégalité devant la mort d'après les statistiques de la Ville de Paris. *Rev. d'Econ. pol.*, Nr. 3 and 4, 1920, 54 pp.

immediately shown by Stevenson,[5] who came forward with similar statistics to those of Hersch, and showed that London exhibited no such picture at all as that presented in the crude figures for Paris. As a matter of fact, both of these papers are on the same weak footing methodologically. They consist merely of the setting of mortality figures against *one other variable only*, and inferring a causal relation from parallelism in variation. This is a scientifically dangerous pastime. The only careful and thorough study of the correlation between poverty and rate of infant mortality that has yet been made by modern statistical methods, that of Greenwood and Brown,[6] shows that when the birth-rate and the artificial feeding rate are held constant, differences in the infant death-rate are not sufficiently influenced or determined by differences in the poverty rate to lead to a net correlation coefficient significantly different from zero.

Another example to the same point may be mentioned. It is a generally held conclusion of the older vital statisticians that there is a high positive correlation between rate of mortality and density of population. A great deal more exact work needs to be done than has been upon this extremely complicated problem, but in the meantime it may be pointed out that in a series of extensive studies, by the method of partial correlation, which have been carried out in my laboratory and will in the course of time be published, it has been found that, within the range of population densities represented in large American cities, there is practically no correlation whatever between density of population and the death-rate from all causes or from tuberculosis, when other variable conditions significantly affecting the populations are held constant. Again the simple statistical parallelism between mortality and one other variable, to the neglect of all others, has apparently led the vital statistician of the old school into a conclusion at least partly erroneous.

4. All this leads me to a fourth, and major, pitfall in the statistical evaluation of public-health activities. This is a corollary to the one we have just been discussing. It is that in all statistical work the probability of drawing conclusions which are in part or wholly wrong is generally inversely proportional to the number of variables taken into account in the computations and reasoning. Such complicated natural phenomena as the rate of mortality are not usually solely determined by a single cause. A number of variables affect the result. One of the greatest advances that has ever been made in the calculus of statistics was the discovery of the method

[5] Stevenson, T. H. C., The incidence of mortality upon the rich and poor districts of Paris and London, *Jour. Roy. Stat. Soc.*, vol. 34, pp. 90–99, 1921.

[6] Greenwood, M., and Brown, J. W., An examination of some factors influencing the rate of infant mortality, *Jour. Hyg.*, vol. 12, 5–45, 1912.

of partial or net correlation. This method enables us to take account of more than two associated variables together.

May I close with an illustration, which may serve as a warning of the dangers of the statistical method? In 1881, before the diphtheria bacillus had been discovered, there appeared in one of the leading medical journals of Germany, by an author[7] of standing, a paper apparently proving, or at least making apparently highly probable the conclusion that the eating of potatoes was the cause of diphtheria! It was shown, by conventional statistical reasoning, that the disease diphtheria first appeared in Europe near the close of the sixteenth century; that it appeared *after* potatoes were introduced as an article of diet; that the disease had increased concomitantly with, and at about the same rate as the increase in the consumption of potatoes as food; that the most severe epidemics of diphtheria occurred at the time of the planting and the harvesting of potatoes; children from two to three years of age were particularly attacked because they played with the potatoes at the time of sowing and harvesting, while the older children were in schools; in one region, Schmalkalden, diphtheria was rarer than in another, Malstatt-Burbach, otherwise similar, because in the former place potatoes were bought in small quantities and immediately consumed, while in the latter place large quantities were laid in, and the people came more often in contact with spoiled potatoes.

Now the point I wish to make about this tale, which seems so weird and ridiculous, now that we *know* what causes diphtheria, is that the statistical reasoning in the paper cited is every bit as good and as cogent as at least much of the statistical work in the field of public health in this country at the present time. We may be, and I fear too often actually are, making just as egregious spectacles of ourselves in our statistical discussions of tuberculosis, infant mortality, etc., as was the gentleman who proved (?) that potatoes cause diphtheria. I hope that the moral of this true story is plain.

[7] Weber, H., Beitrag zur Aetiologie der Diphtheritis, *Allg. Med. Centr.-Ztg.*, Berlin, Bd. 50, p. 860, 1881.

PART IV
THE POPULATION PROBLEM

CHAPTER XXI

The Population Problem[1]

In the opening chapters of the first edition of his *Essay on the Principle of Population*, T. R. Malthus said: "I think I may fairly make two postulata. First, That food is necessary to the existence of man. Second, That the passion between the sexes is necessary, and will remain nearly in its present state, ... Assuming, then, my postulata as granted, I say, that the power of population is indefinitely greater than the power of the earth to produce subsistence for man." In the century and a quarter since these words were published the evidence not only of their truth but also of their profound wisdom has multiplied manyfold. Malthus could know as facts of experience, when he wrote in 1798, only what was behind him. What was to happen in the nineteenth century he could only conjecture.

Proceeding from the earliest to one of the most recent scientific studies of the problem of world population, it is to be noted that Griffith Taylor, in his original and thoughtful paper on "The distribution of future white settlement" in the July, 1922, number of the *Geographical Review* envisages clearly the coming limitation of the earth's population, by reason of its saturation in respect of means of subsistence. The time factor in this population saturation, is one about which there may be difference of opinion. But it really matters little whether one takes Knibbs's estimate of four hundred and fifty years quoted by Taylor (page 402) or that author's own suggested figure of about two centuries or the perhaps even shorter estimate to which my own mathematical studies have tentatively led me. The important consideration is that population, so far as we know, always has grown and certainly is now growing at a rate which, if continued, will some time completely populate the habitable portions of the earth with a density which will be the maximum consistent with the continued existence of human beings.

Figure 95 gives a picture of some aspects, at least, of what has happened since the beginning of the nineteenth century. It will pay us to examine it with some care, because it presents the problem that worried Malthus, as it exists here and now. It will appear with startling emphasis, that nothing which has happened since 1798 has in the least degree mitigated or softened

[1] This chapter is a reprint of a paper with the same title published in *The Geograph. Rev.*, vol. 12, pp. 636–645, 1922.

or altered in any true sense the relentless insistence of Malthus's logic. On the contrary, the developments of the last century have made it far plainer even than it was to so clear-visioned a major prophet as he was, that the population of the world cannot go on increasing at anything like the rate of growth that has prevailed in the past more than a short time longer.

This diagram is constructed from official figures, conveniently compiled in the annual volumes of the *Statistical Abstract of the United States*. It is made according to the following plan. The absolute values for population and for the several other items of production, trade, and the like, included in the diagram, were taken as of value 100 in the year 1800, and then at each subsequent point on the diagram the absolute figures are reduced to relative values on the basis of the 1800 figure taken as 100. The only exception to this procedure is in the case of railways and telegraphs, which had not come into existence in a statistically significant sense in 1800. In those cases, the figures of their first appearance in the statistics (1830 in the case of railways, and 1850 in the case of telegraphs) are taken as 100, and the subsequent values are referred to the initial value as a base. The relative figures so obtained were then plotted on a logarithmic scale. The virtues of such a scale of plotting are, in the present case, twofold. In the first place, it enables us to include in one diagram of moderate dimensions the enormous range of variation exhibited by certain of the items. In the second place, it is a property of logarithmic charts that the *slopes* or trends of all lines are directly and visually comparable one with another. This is not true of charts plotted on an arithmetic scale, unless the absolute values plotted chance to be of approximately the same magnitude. The figures on the left hand margin of the diagram are to be interpreted in this way. If any relative value rises to the line marked 1000, it means that the item exhibiting this figure has increased tenfold. If it rises to the line 10,000, it means that the item has increased, in the period covered, a hundredfold; if to the 100,000 line, it has increased a thousandfold.

The original data upon which the diagram is based, together with the calculated relative values, are presented in table 164. It is to be noted, as a matter of course, that the figures of table 164 cannot possibly be meticulously accurate. They represent, in some of the items, only rough statistical estimates. But they cannot possibly be so far in error as to affect sensibly the general lesson which the diagram teaches.

THE WORLD'S RELATIVE PROGRESS IN POPULATION AND PRODUCTION

With these explanations of the construction of the diagram, we may proceed to see what it means. In the first place it appears that the world's

TABLE 164

World's development of population, production, vessel tonnage, and commerce: 1800 to 1918

YEAR	POPULATION		COMMERCE, TOTAL		TONNAGE, SAIL AND STEAM		RAILWAYS		TELEGRAPHS		PRODUCTION OF					
											Cotton		Coal		Pig iron	
	Absolute, millions	Relative	Absolute, millions	Relative	Absolute	Relative	Absolute, thousand miles	Relative	Absolute, thousand miles	Relative	Absolute, million pounds	Relative	Absolute, million short tons	Relative	Absolute, million tons	Relative
1800	640	100	1,479	100	4,026	100					520	100	11.6	100	0.8	100
1820	780	112	1,659	112	5,834	145					630	121	17.2	148	1.0	125
1830	847	132	1,981	134	7,211	179	0.2	100			820	158	25.1	216	1.8	225
1840	950	148	2,789	189	9,380	233	5.4	2,700			1,310	252	44.8	386	2.7	337
1850	1,075	168	4,049	274	12,334	306	24.0	12,000	5	100	1,435	276	81.4	702	4.7	587
1860	1,205	188	7,246	490	16,600	412	67.4	33,700	100	2,000	2,551	491	142.3	1,227	7.2	900
1870	1,310	205	10,663	721	15,940	396	139.9	69,950	281	5,620	2,775	534	213.4	1,840	11.9	1,487
1880	1,439	225	14,761	998	20,280	504	224.9	112,450	440	8,800	3,601	693	340.0	2,931	18.0	2,250
1890	1,488	233	17,519	1,185	17,461	434	390.0	195,000	768	15,360	5,600	1,077	466.0	4,017	27.2	3,400
1900	1,543	241	20,105	1,359	20,531	510	500.0	250,000	1,180	23,600	6,247	1,201	800.0	6,897	40.4	5,050
1906	1,579	247	27,148	1,854	25,522	634	564.0	282,000	1,200	24,000	7,650	1,471	885.0	7,629	58.7	7,337
1910	1,616	253	33,634	2,274	26,670	662	637.0	318,500	1,307	26,140	9,013	1,733	1,141.6	9,841	65.8	8,225
1911	1,630	255	35,909	2,428	28,298	703	666.0	333,000	1,356	27,120	10,634	2,045	1,309.6	11,290	62.4	7,800
1912	1,643	257	39,570	2,675	29,061	722	683.4	341,700	1,400	28,000	10,301	1,981	1,377.0	11,871	72.8	9,100
1913	1,652	258	40,420	2,733	30,408	755	690.2	345,100	1,462	29,240	10,809	2,079	1,478.0	12,741	77.9	9,737
1914	1,661	260	37,760	2,553	31,674	787	703.5	351,750	1,489	29,780	11,933	2,295	1,346.0	11,603	56.8	7,100
1915	1,672	261	31,302	2,116	31,693	787	717.5	358,750	1,526	30,520	8,805	1,693	1,169.6	10,083	59.0	7,375
1916	1,692	264	46,523	3,146	31,293	777	720.3	360,150	1,564	31,280	9,047	1,740	1,242.9	10,715	71.4	8,925
1917	1,693	265	52,781	3,569	29,805	740	729.8	364,900	1,568	31,360	8,705	1,674	1,317.9	11,361	64.6	8,075
1918	1,699	265	62,802	4,246	31,139	773	732.8	366,400	1,586	31,720	8,845	1,701			62.0	7,750

population has increased in the century a little less than two and a half times. This is, in itself, not an extraordinary figure and would appear to be nothing to be in any way alarmed about. It is far smaller than the rate of population increase under special circumstances, where the population of a particularly favored area may double itself in fifteen to twenty years.

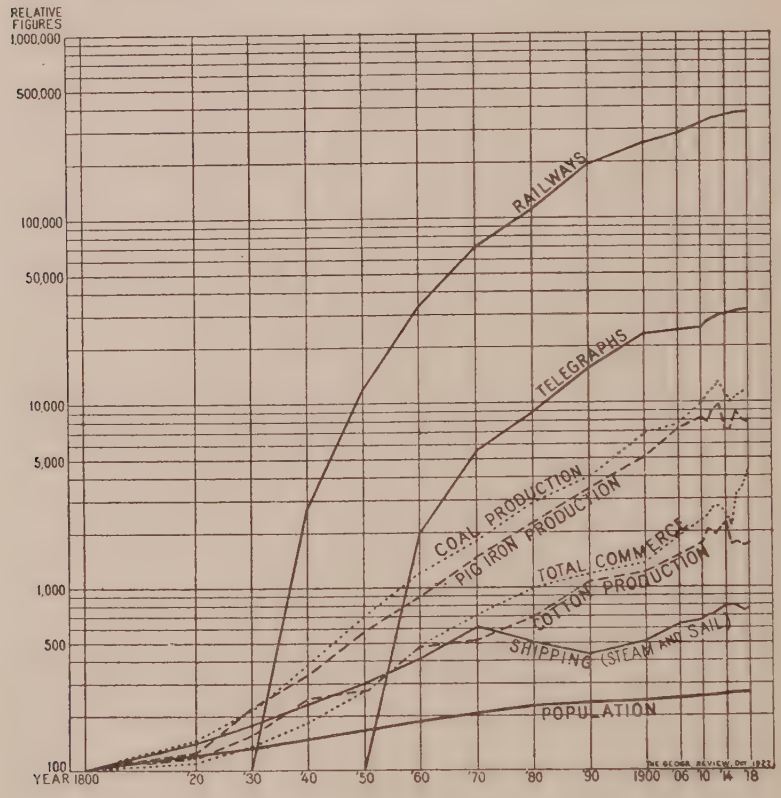

Fig. 95. Diagram Showing the Progress of the World's Population and Production of Certain Materials in the Nineteenth Century

The point of real interest in the diagram is that *the slope of every other line on the chart is far steeper than that of the population line.* Thus it appears that, while the population was increasing two and a half times, the world's coal production and pig iron production increased from fifty to seventy times and by 1917 had increased nearly 100 fold over the conditions of 1800. The world's cotton production increased in the same period twentyfold; the

world's commerce, a little more than that; the world's shipping increased something like eightfold, while the railway mileage increased, in the period from 1830 to 1917, by roughly 3000 fold, and the telegraphs 300 fold.

In the face of such figures as these, one may well ask, "Where are we going?" No sensible person will, for a moment, suppose that such lines of this diagram as those for cotton production, coal production, or pig iron production can go on indefinitely increasing at the rate shown in the 117 years covered. It would be manifestly absurd. Yet, on the other hand, it must be remembered that these figures are world figures, approximate if one pleases, but on the whole certainly not far from the true, unknown, facts. This consideration means that the population of the world, in one way or another, has used up in the business of carrying on its life all of the coal, all of the pig iron, and all of the cotton for which the production trends are indicated on the diagram. There is no such thing as "interworld" trade. Therefore it cannot be asserted that a part of this production went for export and consequently need not be worried about. Trade relations have still to be established with the other planets. When we look at the facts of *world* production, we are also, and implicitly, setting before ourselves the data of *world* consumption, using consumption in the broad sense of usage plus wastage.

Now it appears equally to be true that the whole fiber of our social organization is bound up with the consumption of about the indicated amounts of products. In other words, so much coal and so much pig iron and so much cotton were produced in the last century because the world's people needed them, or, at least, thought they needed them to maintain and continue their lives, individually and collectively, in the way they were doing and presumably wanted to do. Put in another way, there seems no escape from the conclusion that the present stage of the development of the world's civilization requires about the ratios of production to population shown on the chart in order to maintain itself and continue at its present standards of living.

But there at once follows a most significant corollary from this. If it be granted that the coal and pig iron lines, for example, cannot by any possibility be projected for another century or two along the same slopes that their production has followed during the past century, then it must be that, unless the rate of increase of population slows up, the ratio between the production of these goods and the population which has been maintained in the past cannot possibly be continued in the future.

There may be those inclined to interpret the diagram in a directly opposite way; who will say that, since a given population is now able to produce,

owing to all the developments of knowledge which have occurred, such vastly greater quantities of all these necessities than they were able to in 1800, there appears no reason why the process should not go on at the same rate in the next century that it has in the one just past, it being presumed that mankind will increase in the organized intelligence and awareness which constitutes applied science. This view overlooks the fact that besides the human element involved in production, there is another of even greater importance which finally limits the process. It is, that the volume and the surface of the planet on which we live are strictly fixed quantities. This fact sets a limit, if no other does, to the indefinite projection of these straight lines of production trend which make angles of 30 to 45 degrees with the horizontal. For example, one would not have to project the coal production line, along the course it has followed during the nineteenth century, over a very long time before it would reach a point where it would indicate a produced tonnage such that the entire globe would have to be solid coal to permit of its realization. Eventually there must come a turn in these production lines. One suspects that it may have already come in the case of pig iron and cotton.

The curves for railways and telegraphs are interesting in this connection. On the whole, the world has finished the major portion of its railway building. There will, of course, be many more miles of railway built as time goes on; but the additions will clearly be at a much slower rate than has been the case from 1830 up to the present time. In other words, the world is nearing a stage of saturation in respect of railways. It has, roughly speaking, about all it needs.

TRENDS OF THE WORLD'S FOOD PRODUCTION

So far nothing has been said about food, the most important single item in human subsistence. We come here upon much less secure ground, statistically speaking, because there are no particularly accurate world figures for even the most important of crops. The data that are available are distinctly rough estimates, except in the case of sugar, where, owing to the nature of the manufacturing processes involved, the total production is rather accurately known. For the other crops the estimates are, in general, simply the summed returns from countries which have a system of agricultural statistics. Probably this leaves out a significant proportion of the total production. But there is every reason to believe that the *trends* shown by the figures which are available are substantially in accord with what would be shown if accurate total figures were at hand. Inasmuch as it is only the general trend of the figures that we are interested in for the

present purposes, the data set forth in table 165 will suffice. The data are shown graphically in figure 96.

From figure 96 it appears that a process is going on in world food production which is precisely the same in kind, though different in degree, as is shown in figure 95 for the production of other things than food. Until 1916 the food production curves were rising *at a more rapid rate* than the population curve. In the last three years of the chart the upsetting conditions of the war, combined with unfavorable seasons in many regions, caused an abrupt fall in the world's food production. But, leaving these years out of account, it is plain that the world's food consumption (for here as before production means consumption, in the broad sense, when we take the

TABLE 165

World production of principal food crops

YEAR	CORN	WHEAT	OATS	BARLEY	RYE	POTATOES	SUGAR
	million bushels	*million bushels*	*million bushels*	*million bushels*	*million bushels*	*million bushels*	*million pounds*
1895	2,835	2,593	3,008	916	1,468		17,779
1900	2,793	2,641	3,166	960	1,558	4,382	19,370
1905	3,461	3,327	3,510	1,180	1,496	5,255	21,310
1910	4,032	3,575	4,182	1,389	1,673	5,275	33,415
1911	3,461	3,541	3,786	1,375	1,579	4,749	38,083
1912	4,055	3,760	4,585	1,457	1,901	5,873	35,585
1913	3,587	4,127	4,697	1,650	1,880	5,803	40,788
1914	3,878	3,486	4,035	1,463	1,597		41,972
1915	4,213	4,173	4,389	1,560	1,586	3,044	41,512
1916	3,101	2,279	3,941	1,437	530	1,720	37,069
1917	3,483	2,224	2,975	914	434	2,734	37,729
1918	3,038	2,818	3,052	1,077	529		38,375

world as a whole) has increased faster in the last twenty years than has population. This is particularly marked in the case of the commodity sugar, which in some degree may be regarded as a food luxury.

It will be understood, of course, that not the whole of any one of the crops shown in figure 96, is *directly* consumed as human food. In some cases, notably corn, the fraction that is thus directly consumed by human beings is relatively small. But indirectly and in converted form, through animals and manufactured products, the bulk of all edible farm crops contributes in greater or less degree to human nutrition. We eat the corn in the form of pork, we drink (or did drink) a good deal of barley as beer, we eat some of the oats as oatmeal, some as eggs, some as milk, and so on.

Now, a superficial view of the situation revealed in figures 95 and 96 is likely to be extremely complacent. Population has grown since Malthus's day. Yes, about as he said it would. But Malthus quite overlooked the powers of man himself to make nature do his will. As the population has increased man has made the means of subsistence increase still faster. So far from population catching up with the means of subsistence, it is plainly out-distanced in the race. So will the unthinking interpret our charts.

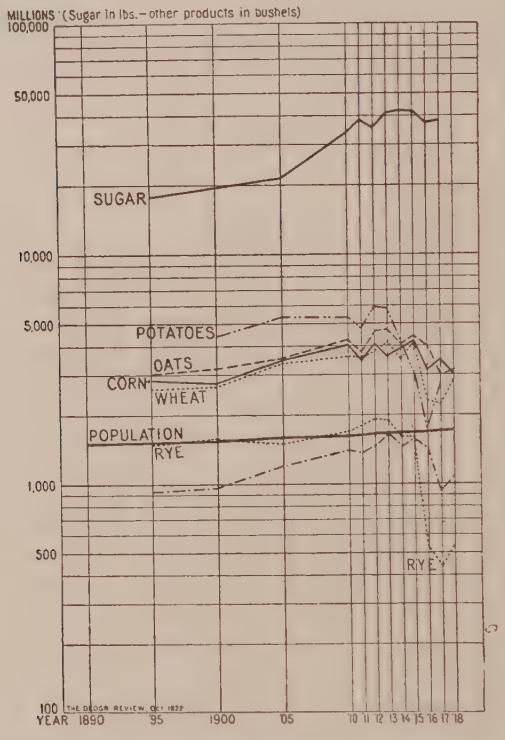

Fig. 96. Diagram Showing Progress in World Food Production in Relation to Population

But we are unable to convince ourselves that such complacency has any warrant. We believe that anyone who will take the trouble to read even the present chapter carefully and will ponder over the *facts* it presents, rejecting if he likes everything that savors of opinion or theory, will be bound to feel some misgivings about the world's ability to go on indefinitely increasing both its population and its average standard of consumption

or of living. For precisely that is what we are now doing. The steeper slopes of the production as compared with the population lines can only mean (recalling always that we are dealing with the world figures) that the ratio of consumption to population has been steadily widening. But a widened consumption ratio represents a more extravagant standard of living. It can, broadly speaking, mean nothing else. If people, taken at random, consume (use and waste) more goods now than people did say twenty years ago, then plainly their standard of consumption has gone up.

RESULTS OF OTHER STUDIES

Space will not permit any exhaustive review of the literature, but I desire to quote briefly from two recent writers on the general subject of population, in order to show that I do not stand alone in the views suggested. One of the most careful and thorough of recent students of the problem is Thompson. In rounding up the results of his study he says:

> Another conclusion which seems to me to be warranted is that population cannot continue to increase at its present rate without being more and more subjected to the actual want of food, provided the distribution of labor between agriculture and the non-agricultural industries continues in its present trend (the trend found in the more highly developed countries). Nor can a greater and greater proportion of the population be devoted to agriculture and the present rate of increase continue without checking a progressive standard of living. The non-agricultural industries are not yielding increasing returns in such ratio that they can furnish the necessary material means for a progressive standard to such a rapidly increasing population. Thus whatever the direction of development, a progressive standard of life and a population increasing from 1.5 to 2.0 per cent a year cannot go on together for long, in a large part of the world. Therefore, either our present standard of living must be simplified as an increasing proportion of the population becomes rural or the present rate of increase of population must be lowered. Probably both must take place in order to have a really progressive civilization.
>
> To simplify our present standards of living does not necessarily mean a lowering of them. It means rather that a good many of the things of our civilization which we consider essential today may be found to be merely passing phases, induced by our rapid industrial development. We have become accustomed to think of civilization and culture and progress as of necessity involving all the complexities of our present existence. It is open to question, however, whether much of our present complexity is not a hindrance to real culture rather than an aid. There cannot be much doubt that as people are becoming more educated they are becoming more self-contained, and they begin to see that the way to get the most out of life is to put the most into it, and not to surround themselves with all the luxuries and baubles they can afford.
>
> But it is doubtful whether even such a simplification of life of the people of the more highly civilized nations will enable them to support in comparative comfort an ever-increasing proportion of their populations, or even the same proportion as at present, if it is not accompanied by a lower rate of natural increase of population. A slower rate of increase of population will give more time to adjust standards of life to surrounding condi-

tions and to direct the course of progress; without it most of our efforts must be directed towards the more pressing of the problems of our present-day life. A greater and greater control over the growth of population is essential to a growth of rational social control.[2]

Another leading student of the problem, East, has this to say in a recent paper:

Let us think in terms of land. A careful study of the available statistics shows that, by and large, it takes about 2½ acres to support each individual. Some self-supporting nations can make out with less, but only when they put all their efforts into cultivating their richest land; others with less efficient methods take much more. Taking this as the average figure, then, it is necessary to plow, plant, cultivate, or otherwise tend, some 37 million acres more land each year than was ever so treated before, if the nations of the earth are not to go hungry.

It is a curious illustration of the general tendency of people to be optimistic about everything, that even many of our most eminent economists believe this wresting of additional food from the soil to be an easy task. They draw wonderful pictures of what science has done in the past few years, make roseate prophecies of similar advances in the future, and keep everyone happy with the idea that the human race has unlimited credit on Nature's bank. Never was logic more fallacious. The total land area of the globe, 33,000 million acres, seems large, it is true, but one cannot utilize all this area for raising crops. Take out the mountains, the deserts, the undrainable swamps, in short the areas not available for agriculture, and there is left about 13,000 million acres. Of this potential world farm some 5000 million acres are now being cared for by the hand of man. According to our previous calculations, then, the maximum population the earth can support is a little over 5000 millions. And here is the heart of the matter—the time when this important event would take place at the present rate of increase is not so far distant but that some of our grandchildren would live to see it. Mind, I do not say the world is going to fill up and run over like a tub within the next century or so. Of course it is not. The difficulties of digging out a living, the positive checks to population, will see to it that the present rate of increase diminishes, even if no other checks intervene. I am merely showing the meaning of this speed.

In the second place, human genius is not doing for agriculture just what our economists seem to think. The industrialization of the Caucasian world which has gone on at such a rapid pace during the last fifty years has provided for more people, it is true. In fact, population increase, though rapidly rising, has not really caught up with the increase in production made possible by the multitude of mechanical inventions. But why has this come about? There is one reason and only one reason for the situation. We have had a reserve of new land to draw upon. Mechanical invention simply made it possible for a given unit of man power to cultivate more land and to distribute its products more rapidly and equitably. With the exception of the credit to be given the mechanical production of chemical fertilizers, thus far negligible in a world-sense, the Age of Steel has not aided agriculture one iota, when computation is made on unit area. In other words, the provisions John Doe raises on his hundred-acre farm have not been increased because of traction plows and steam threshers. But John Doe has had cheap land available and

[2] W. S. Thompson, Population: A study in Malthusianism (*Columbia Univ. Studies in Hist., Econ. and Pub. Law*, vol. 73, no. 3), New York, 1915, pp. 163–165.

has been able to cultivate more of it than he could by the old hand methods. Yields per acre were actually decreased. What will happen when the new land gives out? It is giving out, giving out rapidly. Well the result will simply be this: the machine farmer will give way to the hand farmer. The American who now cultivates 26 acres per capita will have to put more man power into his efforts and will approach the Belgian standard of 5 acres per capita. He will get greater yields on a given acre by so doing, even as the Belgian farmer gets greater yields, but the yield per capita will sink slowly downward. So few people seem to appreciate this difference of standard. They look at the English yields of wheat and German yields of potatoes and wonder why the yields per acre in the United States are so low. They are low simply because the United States has had new lands to exploit. The country has been in an era of "increasing returns;" when shallow plowing, incomplete cultivation, and other cheap extensive methods would still yield a fair profit. European agriculture, on the other hand, has long been experiencing the pain of "decreasing returns." Year by year they have been faced with the necessity of getting more and more out of their land, to pay the mounting rentals. They have succeeded remarkably well; but they have only had this success because they have put more toil, more man power into their work. We are now entering on this era of diminishing returns per person in this counrry. Our yields per acre are rising and will continue to rise. But they will rise because of the increased effort put into the work rather than because of new methods of tilling and of time-saving devices.[3]

These quotations from two of the leading investigators of the problem of population at the present time will be sufficient, it is thought, to indicate that the author is not alone in supposing that this is a real problem, perhaps the most significant one facing mankind today.

[3] E. M. East, Future food prospects, *World Agric.*, vol 2, 1921, pp. 130–132.

CHAPTER XXII

Biology and War[1]

Science is playing a part in the conduct of the present world war far beyond anything ever dreamed of as a possibility before its beginning. The physicist and the chemist have been called into consultation with regard to practically every sort of military activity, both offensive and defensive. They have been asked on the one hand, to devise new mechanisms of destruction, and on the other hand to provide effective means of defense against such measures of annihilation as the enemy has been able to put into operation. The response to these demands has been generous, timely, and effective in all of the countries at war. In view of his contributions in these directions the university professor of physics or chemistry seems in a fair way to attain, when the war is over, a position of respectability and esteem in the world's affairs never before imagined in his wildest dreams. The submarine, the aeroplane, gas warfare, as indeed practically all of the new fighting methods which have been put into operation in the last few years, are highly recondite developments of physical science, using the term in a broad sense to include chemistry, mathematics, and even astronomy.

One has heard very little about the immediate help rendered by biology in the conduct of the war, except in relation to the medical sciences, where the contribution is directly to the salvaging of the human wreckage with which the pathway of war is strewn, and only rather indirectly towards its winning. It is generally taken for granted, and to a considerable extent even by professional biologists, that in the nature of things the biological sciences, other than the medical, can have only rather a remote and indirect relation to the conduct of war.

[1] The material in this chapter was first delivered as a lecture before the Washington Academy of Sciences on May 9, 1918, and subsequently published under the same title in the *Jour. Wash. Acad. Sci.*, vol. 8, pp. 341–360, 1918. Rereading it five years after the termination of the exciting period in which it was produced reveals surprisingly few things which I have any desire to change. It has therefore seemed best to leave it in practically the same words. Perhaps I am deluded but I still believe that the analysis of some of the biological antecedents and consequences of war is essentially sound, and has significant bearing upon not a few of the post-war troubles which are so sorely vexing men, particularly those living in or near the countries of the European combatants. The reason for placing this chapter in its present position in this book is because of its bearing upon what used to be considered one of the most effective checks to population growth, a great war.

One purpose of this discussion is to make some examination of the biological philosophy of war. It has seemed to me that if one does this, he is likely to come to the conclusion that the ordinary valuation of the relative significance of the physical and chemical problems connected with war as compared with the biological problems is substantially the reverse of the true valuation. To begin with, we should remind ourselves of a distinction which is often forgotten when one attempts to evaluate in military terms the potential contributions of the different sciences to war. Essentially what the physicist and the chemist contribute is toward the creation, development, or perfection of some destructive or protective *mechanism*—at best an inanimate, impersonal machine. But the very essence of a fight is that it is between *living* things. A 120-kilometer gun, or a submarine, or a tank, cannot of and by itself make war. All such engines of destruction are only the secondary implements of war. The primary implements are biological entities—men. Without these entities there neither would nor could be any war. So then, obviously, the primary problems of war are biological problems. They are such problems as why men fight; what kinds of men make the best fighters; what conditions, both internal and external, biological and environmental, conduce to the most effective fighting; and what are the probable biological consequences (including physiological, social, and genetic) of the fight, both to the winner and the loser. These are the sort of problems to which the biological sciences can alone make any significant contribution and they are clearly much more fundamental than those entailed in the designing of a new aeroplane or submarine.

Furthermore, it admits of no doubt that the accumulated knowledge in the field of biology could be utilized in a way to be of large strategic value. The biological analysis of the events of the war as they pass might be made of direct military importance in the forecasting of the future course of events. An illustration here is found in what has happened in Russia. The collapse of Russia was at bottom not due to any shortage of powder or shot or other secondary requirements of military activity, but it came essentially because a Russian is, in certain respects, a totally different kind of animal from an Englishman, a Frenchman, an Italian, or an American. Because he is a different kind of animal he has throughout his past history reacted to certain sorts of stimuli in a different way than would or did the individual of the other nations mentioned. Any thoughtful student of the biological aspects of history—that neglected branch of science which Frederick Adams Woods has been trying for years to interest people in, under the somewhat forbidding label "historiometry"—could have foretold with considerable precision both as to time and event, or better eventuality, just what Russia's contribution to the cause of the Allies would be.

What I have so far said will serve as a general indication, I hope, of the fact that we have signally failed to make effective use of the contribution to war that biological science, in the broadest sense, is potentially able to make. The indictment here, if there be any, falls upon the class to which I professionally belong. What I wish to do in the remainder of the time at my disposal is to discuss a few of the more important biological problems of war, in the hope that such discussion may serve in some slight degree at least to arouse interest in these problems on the part of many biologists much more capable of dealing with them than I am. If this can in any way be accomplished I feel that biology will make to the cause in which we are all vitally interested a contribution second to none.

WHY MEN FIGHT

War constitutes a gigantic experiment in human evolution. For the experimental study of evolution in lower organisms we have many laboratories and institutes. In such laboratories one studies the effect on the race of modifications in the environment, of crossing the different races, and of various other factors, which may be supposed to have a determinative influence in bringing about evolutionary change or modification. A great war performs all these experiments on a stupendous scale with the human organism as material.

In saying this I am not at the moment referring to the relation of natural selection to war. That is a topic to which I shall come later. I am here referring to a very much broader aspect of the question. War is not merely selective (if it be so at all) through elimination by death of men at the front. Its biological effect on the human species is much more profound than anything which could possibly result from any merely selective process. War makes a most complete and far-reaching change in the whole biological environment of the human beings of the countries engaged in it, and if the number of these countries is sufficiently large it affects the whole world. In this regard, it is most nearly comparable to what the geologist calls a catastrophic change in evolutionary history. The reason why war induces so profound a change in human environment is that it disturbs every psychological and social relation of men with each other. For modern civilized man the environment does not mean primarily the climate, the flora, or the geological structure of the place in which he lives. To a very considerable extent civilized man controls and modifies the impingement of the direct physical elements in his environment. The important elements of human environment are those which grow out of the activities of the human mind, or as one may broadly say, the psychological and social elements.

These include all the social relations which are built up during years of peace. But war, in and of itself, brings about an entirely new revaluation of all existing social, economic, intellectual, and moral relations. This is true not alone for the combatants, but for all the non-combatant or neutral nations. In a war such as the present one men everywhere begin to reconsider their thought and action about such things as what constitutes proper education for their children, what is a desirable mode of activity for the church, what sort of activities in the conduct of business may be tolerated, and a thousand other of the complex and manifold relations between human beings.

True evolutionary change in a strict philosophical sense means a definite and permanent alteration in a group of organisms, both in the group as a whole and in the individuals composing it, as individuals. When one uses the term "permanent" in this connection it should, of course, be understood always to carry the qualification, permanent until the conditions which produced the initial evolutionary change themselves become altered. Now human social evolutionary change rests upon two broad general bases instead of the one upon which the organic evolution of lower forms of life depends. Lack of recognition of this fact has been a fruitful source of failure to arrive at philosophically sound conclusions in many discussions of the social evolution of man, undertaken from the biological point of view.

The basic element and limiting factor in organic evolution is the germ plasm. It is at once the race stabilizer and the race initiator. The germ plasm is the physical basis of inheritance in general. Borne in the reproductive cells of the organism it is the one thing which preserves physical continuity between successive generations of organisms. If successive generations are to differ from one another biologically there must be concomitant and equivalent changes in the germ plasm. Genetic and eugenic research has abundantly proven that the germ plasm plays the same rôle in human inheritance and human evolutionary changes that it does in lower organisms. Here one needs only to mention the studies of Galton, Pearson, and Davenport by way of illustration. Many others might be added to the list.

Besides this strictly biological base of the germ plasm there is also another underlying factor in human social evolution which is nearly, if not quite, of as great significance. I refer to that complex of ideas and actions which has been rather badly called "social inheritance." This factor operates in somewhat the following manner. Starting from a germ-plasmic base the individuals composing any social group are biologically differentiated from those forming other social groups. On this account they develop social

relations and social institutions of a sort in some degree unique and peculiar to the group. Once started, these social relations and institutions acquire a sort of inertia which in and of itself tends to stabilize them quite without any conscious activity looking towards stabilization on the part of any of the component individuals in the group. This inertia extends within the group in an extraordinary degree to every sort of social relation, including even the minor conventions. It makes the whole social fabric, which, as we have seen, constitutes a very important element of human environment, extremely resistant to change or alteration of any sort. Ordinary social forces produce but little effect. It requires years of unremitting effort to bring about even mild and minor social reforms or changes in the ordinary normal course of human events. It has taken nearly seventy-five years to get as far forward as we are with the prohibition movement in this country. More strongly socially inherited institutions would be still more difficult to alter. To illustrate the point, let us consider the social-economic institution of interest. It is entirely possible, not to say easy, to conceive a society so organized that credit and the interchange of credit would be effected without the institution of interest. But try to conceive the concrete possibility of putting into actual operation in the civilized world today a system which would do away with interest charges. The mind balks at the thought. The inertia of this institution, its social inheritance, is so strong that to change it would be a task of commensurate relative magnitude somewhat approaching to the task of so changing the germ plasm of the human race that man would have, for example, no vermiform appendix. Both are extremely stable things which cannot be easily or quickly changed by the operation of ordinary forces. Both changes involve an alteration in stably equilibrated systems, and it is a general characteristic of such systems that they do not change either frequently or easily. The inertia of social relations, which is I think a better term than social inheritance, is simply a special case of the general phenomenon of the natural occurrence of systems in stable equilibrium, the manifestations of which in the inorganic world have been so brilliantly expounded by Lawrence J. Henderson in his book *The Order of Nature*. It is precisely homologous to germ-plasmic inheritance in the biological realm and not less potent in its influence as a stabilizing factor in human social evolution.

The one outstanding cause in present-day civilization which can quickly break the inertia of social institutions and induce changes, and by so doing perform a function in the scheme of social inheritance analogous to that of mutation in physical inheritance, is war. It operates to direct sharp and searching attention to the real significance of every social institu-

tion, from the standpoint of national efficiency, national economy, and national well-being. If under such stressed examination change or reform appears to be necessary it rather quickly follows. The inertia of the long established is broken by the conditions of war.

If it would not take us so far afield into philosophy and perhaps even metaphysics I should like to pursue this point further, but I think that perhaps enough has been said to make clear the only thing which is requisite here, which is that if we are profitably to discuss the biological philosophy of war we must recognize that besides the influence of the germ plasm in human affairs we have to deal with another general factor of a social but still essentially biological character, namely the inertia of social relations and institutions themselves, which stabilizes them against sudden or rapid alteration by any but the most catastrophic causes such as great wars.

As a concrete example of the application of what we have been discussing to present conditions, we may take the case of England. Already since the beginning of the war England has passed and ended a stage in its social evolution to which it can never return. The institutions and people of that country in all their outlook on social relations in the widest sense have been essentially and fundamentally changed, and however the war may end, will be permanently different from what they were five years ago. Anyone who will take the trouble to read the recently promulgated program of the English labor party will realize how profound the alteration has been. Or, again, consider the whole history of the Home Rule question. More progress has been made towards its solution since the beginning of the war than in all the previous struggles with it.

To bring about such changes, which constitute a real and definite step in social evolution, it is not at all necessary that the enemy should win a war. It is war itself which accomplishes these alterations in human relations and human beings. It only need be sufficiently comprehensive in its magnitude, and sufficiently long continued in time, to produce definite and permanent evolutionary changes through alterations of social relations and institutions.

There is a further side to the evolutionary aspect of war which we have not yet considered. If we view the matter in terms of nations, not of individuals, it is at once apparent that war is a deliberately planned struggle between biologically unlike groups of individuals for the purpose of maintaining or bettering their status in the general hierarchy of group domination or precedence. A modern war is not entered into casually and without some degree of both spiritual and material preparation. In the nature of the thing itself it cannot be so entered. To make a whole nation want to fight, including all the ignorant, because uninformed, people in it, it is

necessary that their emotions be stirred, either by some act or supposed act of an offending nation or else by deliberate emotional propaganda. At the outstart of any war this emotional incentive to belligerency is wholly lacking in a very considerable portion of the populations of the nations involved. It has to be worked up, a process in which the enemy always renders most efficient service, by such things in these latter days as air raids over inoffensive towns, sinking passenger vessels without notice, or in other ways too revolting to mention. Pending the general distribution of rage in the involved populations, the business of war has to be planned and executed by the nation's leaders in as detached and impersonal a manner as any other great business enterprise. This fact, which to a resident of another planet not accustomed to our ways might seem strange, raises two questions: In the first place, why do national leaders enter so coolly, and yet under certain conditions so eagerly, upon such a ghastly business as war; and in the second place, why do the common people not only permit them to do so, but follow them with all their energies when once the business is well under way? Some biological facts will help us to understand the answers to these questions.

The general biological fact of individual variation is, of course, familiar. No two individual animals of any sort, human or other, are precisely alike. Individuals vary or differ among themselves. Of these variations or differences some are superficial and transitory, but, on the other hand, many have a deep-rooted and ineradicable germinal basis. Perhaps the most general result of modern genetics is to show the extent to which variations, often slight in their external manifestations, have a definite germinal basis, reappearing unaltered again and again in the successive generations arising from the same germinal stock. The same fact of variation holds equally true in respect of races and national groups, provided in the latter case they have existed as socially isolated entities sufficiently long for a distinct feeling of nationality to develop. The variation in national groups involves, as in the individual, all sorts of characters, psychological, social, and moral, as well as physical. In new nations, changes in the psychological, social, and moral characters appear and become fixed by the process of social inheritance sooner than in the strictly physical characters. The fact is that the groups of people, which, in political terminology, are called nations, in the great majority of cases become rather quickly biologically differentiated if they are not so from the beginning of their national life. A German is different from a Frenchman or an Englishman or an Italian. These differences are not merely physical. They involve every mental attitude, appetite, and responsibility, which are the factors governing action.

To recognize the fact of biological differentiation or variation is in no sense to assert difference of position in the evolutionary scale. There is no evidence that among these larger and more developed national groups it is proper to speak of one as superior or inferior to another. Philosophically, all such comparisons of races or national groups are untenable, for the reason that they all involve by implication comparison or measurement with some absolute and unique base or yardstick. But no such absolute base of social evolutionary comparison exists. For example, someone might conceivably contend that the Germans were superior to the Hottentots, but it would be a difficult thing to prove in general or absolute terms. Measured by common sense standards one would no doubt find that in some respect—physical, or moral, or even perhaps intellectual—the Hottentot is a relatively better man *in his environment* than the German is in his. Plainly, in order to be just to either the Hottentot or the German each should be measured by a different yardstick. But this quite prevents saying in any absolute terms which of the two is the superior race. Like so many other things "it all depends." But this logical difficulty only makes it all the clearer that Hottentots are *different* from Germans.

Not only are the different races and national groups generally different, but broadly speaking, they all want to stay so, and this is what causes all that special sort of trouble, which is war. The resentment against the high-handed imposition of that Prussian "Kultur" which we are all so strenuously opposing, arises not so much from any logically proved defects in this particular brand of Kultur (though parenthetically one may remark that they appear to be sufficiently numerous), but rather because, being different, the people of other nations simply do not want it. They prefer their own particular brand of thought and action. The one fundamental thing which an Englishman or a Frenchman will fight against to the last ditch is any attempt to make him over into a German.

The same feeling is exemplified in every war. We fought bitterly for it in the Civil War. The people who originally settled in the southern portion of the United States were biologically and socially different in several important particulars from those who settled in the northern part. The Southerner fought hard and well for four years to keep from being dominated by the Northerner. He had a strong feeling, which was to a certain extent justifiable, that domination meant the obliteration, for all practical purposes, of certain differences which had up to that time existed between him and his Northern neighbor. The same feeling was a potent factor in making the Revolution. There was a dawning national consciousness in the colonies which was based upon a beginning of social and biological differ-

entiation. The mother country very unwisely refused to recognize, or foster, or even tolerate these differences. In consequence, she lost her colonies.

In general, why men deliberately plan wars is because they are different biologically, in structure, habits, mental outlook, thought, or other ways, and wish to preserve intact their differentiations. The more truly conscious they become of these group differences, the more likely they are to fight as groups. As soon as they attain the first glimmerings of such consciousness they are apt to see, or to think they see, something in the behavior of their neighbors which threatens the maintenance of that which begins to mark them as a nationality. It is the business of their national leaders to be on the outlook for such things. They may merely fancy that they detect some danger to the maintenance of even their present status in something that a neighboring nation does. It may be a very intangible thing, and the interpretation of its significance may be entirely wrong, but that does not matter. The fighting promptly follows.

But someone will ask: *Why* does fighting follow? Why not arbitration or some other peaceful means of settling what is in many cases, at least, merely a trivial difference at the start? The biological answer is again clear. The human animal, in common with other higher vertebrates, has come to be endowed with emotions, of which rage is a very important one. In the intercourse of men and nations such things as insults, real or fancied, triflings with honor, either individual or national, attempted interference with natural or vested rights, larceny of territory or other goods—all these and similar sorts of activity vastly too numerous to catalog, tend to call forth the emotions of anger or rage. More particularly are acts of the sort mentioned sure to stir the emotions of a people if they are perpetrated by foreigners, those who do not belong to the same group. People of one's own kind may with impunity do things which another kind of people cannot do without exciting very violent emotions. The significant biological fact is that, however induced, the emotion of rage automatically and inevitably causes certain definite bodily changes and activities, as has been demonstrated by the brilliant researches of Dr. Walter B. Cannon, described in his remarkable book entitled *Bodily Changes in Pain, Hunger, Fear and Rage*. The bodily changes induced by rage are precisely those which make the organism ready for fight. They are the visceral preparation for the translation of emotion into action. The researches of Cannon have shown, as he says, "a number of unsuspected ways in which muscular action is made more efficient because of emotional disturbances of the viscera. Every one of the visceral changes that have been noted—the cessation of processes

in the alimentary canal (thus freeing the energy supply for other parts); the shifting of blood from the abdominal organs, whose activities are deferable, to the organs immediately essential to muscular exertion (the lungs, the heart, the central nervous system); the increased vigor of contraction of the heart; the quick abolition of the effects of muscular fatigue; the mobilizing of energy-giving sugar in the circulation—every one of these visceral changes is *directly serviceable in making the organism more effective in the violent display of energy which fear or rage or pain may involve.*" It is clear that we have here a first class reason why men fight. It is, in short, because they get mad at each other. It is fair to say that this has been suspected for some time past. What the physiologist has shown us that we did not know before, however, is the reason why rage is more generally followed by fighting than by judicial arbitration.

No interested person or nation was ever known publicly to allege any such reasons as those just discussed for participating in war. That fact, however, may with safety be taken not to invalidate the point. Most men are human and a liking for the outward trappings of inner grace is a highly human attribute. As war begins, and while it continues, even on to the final ending around the table of the peace conference, everyone involved alleges a wide variety of highly moral reasons as to why he is participating. As a matter of fact, he perfectly well knows, if he is at all intelligent, and at all given to facing the actual facts as they are, that the high principles have absolutely nothing to do with the *reasons* for his fighting. They serve a wholly different and much more useful and admirable purpose, in that they *justify* instead of explaining his belligerency. The explanation of why men fight is very simple. It is, first, because their kind of people is different from other kinds; second, because they want to make sure that their kind shall either maintain or improve its status in the world, and that which is thought to ensure most certainly the maintenance and extension of group differences in the widest sense is relative politico-social domination by the group; and third, because of a general physiological law that certain emotions tend to lead to *action.* So long as men are capable of becoming enraged there is potential danger of a fight.

THE BIOLOGICAL CONSEQUENCES OF WAR

Any discussion of the consequences of war, from a biological standpoint, demands as a first requisite the consideration of natural selection in relation to war, or, as it is perhaps more commonly put, "Darwinism and war." German philosophers of all degrees of attainment have been particularly

addicted to speculation in this field. The view commonly held is that in war we have practically the only existing agency of natural selection operating with full vigor upon the human species. It is contended that when two nations engage in warfare with each other the principle of the survival of the fittest accompanies the operation with all its traditional crudity and finality. No better exposition of this viewpoint can be found than that set forth by my friend and colleague, Vernon Kellogg, in his remarkable book *Headquarters Nights*, from the after-dinner remarks of the distinguished German biologist who figures in the narrative under the name "Professor Von Flussen." Kellogg expounds the philosophy of war after Von Flussen in the following words:

> The creed of the *Allmacht* of a natural selection based on a violent and fatal competitive struggle is the gospel of the German intellectuals; all else is illusion and anathema. The mutual-aid principle is recognized only as restricted to its application within limited groups. For instance, it may and does exist, and to positive biological benefit, within single ant communities, but the different ant kinds fight desperately with each other, the stronger destroying or enslaving the weaker. Similarly, it may exist to advantage within the limits of organized human groups—as those which are ethnographically, nationally, or otherwise, variously delimited. But as with the different ant species, struggle—bitter, ruthless struggle—is the rule among the different human groups.
>
> This struggle not only must go on, for that is the natural law, but it should go on, so that this natural law may work out in its cruel, inevitable way the salvation of the human species. By its salvation is meant its desirable natural evolution. That human group which is in the most advanced evolutionary stage as regards internal organization and form of social relationship is best, and should, for the sake of the species, be preserved at the expense of the less advanced, the less effective. It should win in the struggle for existence and this struggle should occur precisely that the various types may be tested, and the best not only preserved, but put in position to impose its kind of social organization—its *Kultur*—on the others, or alternatively to destroy and replace them.

That this is a fair and typical exposition of the views of German biologic philosophers regarding war will be readily granted without argument by any evolutionist who is familiar with the literature in this field. The principle of natural selection was seized upon by no one with greater avidity than the Germans. The strictly mechanistic features of this doctrine, which Darwin himself seemingly always felt to be a potential source of weakness, were the very things which made the strongest appeal to the Germans. In the hands of Haeckel, and particularly Weismann, natural selection was developed into a complete philosophical system of biology, in which any lack of biological evidence regarding the actual operation in nature of the basic principle was more than compensated for by the wooden finality of the logic.

As years went on the German statesmen and political philosophers became acquainted with the content and possibilities of what their biological con-

freres had by that time come to call with considerable unction "Neo-Darwinism." They presently saw the great possibilities which the principle of natural selection offered in fostering and developing in the minds of the people the militaristic ideal, the will to conquer. For thirty years every German school boy and girl has been taught what natural selection means. This same glorious principle that the fittest alone shall survive, and its converse that the survivor is the fittest, have been the corner stones on which modern Germany has been built. Various remote and far removed causes have been assigned as contributory to the present conflict, but one highly important cause—perhaps in a philosophical sense the most significant of all—has been very generally overlooked. I believe it to be literally true that the one event in the history of Western Europe which more than any other single one laid the foundation for the situation in which Western Europe finds itself today, was the publication in 1859 of a book called *The Origin of Species*. With what horror would that gentlest and kindest of souls, whose mind conceived and executed this work, have been filled could he have foreseen the frightful welter of blood which has resulted from the gross perversion of his views by German biologists.

Let us examine with some care the meaning of natural selection in its relation to war. In the first place, it must be remembered that nowhere in nature does natural selection, as indicated by modern careful study of the subject, operate with anything like that mechanistic precision which the German political philosophy postulates. In a recent paper read before the American Society of Naturalists, I presented a number of examples from the literature illustrative of this point, and I need not repeat them here. Nature often does not operate on the natural selection basis, though logically—at least in formal logic—it ought to. Much less does natural selection operate in a rigid and mechanical manner with reference to human affairs. It is perfectly clear that no war in this day and age is, in any proper sense of the word, literally a struggle for existence. The German people have from the beginning tried to make it appear that the present war is, from their standpoint, exactly this. They have insisted again and again that their national existence, their continued survival as a nation was threatened by their neighbors, but such a view has only to be stated to any fair-minded unbiased person to prove its utter absurdity. Could anyone but a German seriously maintain that the French, or the English, or the Italians, or the Russians, would have wished for, or would have attempted if they could, the annihilation of the German people? Theoretically, such a feeling or desire is conceivable, but practically everyone knows that it did not exist. Normal human beings are simply not constituted that way.

Furthermore, military results are not, in fact, measured in terms of biological survival. History shows that defeated nations survive just as definitely and truly as conquering races or nations. No better example could be found of the fallacy of the completely mechanistic natural selection idea with reference to war than our own Civil War, which was the most severely and bitterly fought of any war in recent history before the present conflict. No question of biological survival was involved at any stage; it was a struggle to effect the survival or elimination of certain politico-social ideas held by one group of people and not by the other. These ideas were slavery and secession. One of the contending groups was defeated; no military decision can ever be more complete and final than was that reached in the Civil War. If military conquests or defeats ever mean biological survival or elimination the principle should have been exemplified in the Civil War. Yet as a matter of fact and of course the defeated group was not eliminated in the biological sense, but biologically survived, and not only survived, but has become as a group more active, more progressive, and more distinctly differentiated biologically than it was before the conflict.

Other wars at other times show the same things. Take the case of peoples subjugated by military conquests; they are not eliminated, but on the contrary they survive, using the word in its strict biological signification. The natives participating in the Indian mutiny suffered a stinging military punishment. Yet today the natives of India survive, and their institutions survive. Again, take another example: it was necessary for us some years ago to conquer in a military sense the Filipinos. The unpleasant task was accomplished in a thorough-going manner. A complete military decision was made, but the Filipinos were not biologically eliminated, and today have a significantly stronger and more real national feeling than probably ever before in their history.

Nearer events prove the same point. No more ruthless attempt at the biological elimination of a nation was ever made than that undertaken by the Germans against Belgium in the summer of 1914 and continued to the present time. Yet, does anyone, even a German, delude himself into the belief that the Belgian people and the Belgian national feeling do not survive today, and will not continue to survive?

The plain fact in the matter is that the proudly ruthless philosophy of Treitschke and Bernhardi is not only immorally cruel, but also immortally stupid. This whole crude and mechanistic view of war as a process of natural selection is really most unbiological in that it takes no account of the most fundamental of human biological characteristics—namely, those which distinctively differentiate man from lower organisms, his mental and

moral qualities. Biologically, nationality rests on the group spirit of the people, which in turn means differentiant variations ineradicably ingrained in their germ plasm. Nationality can only be eliminated in the biological sense by the complete and total destruction of the germ plasm of the people of the nation, because it depends upon things which are to a substantial degree, at least, unchangeably and permanently determined by that germ plasm. Killing a percentage of the male population on the battlefield is as silly as it is a pitifully sad method of attempting to destroy the germ plasm of a nation. What a defeated nation loses in war is simply its status in the international political hierarchy either temporarily or permanently. It suffers, broadly speaking, no fundamental biological loss. The Chinese today, after a century of hopeless military defeats which left them an inert and pacifist nation are just as truly and completely biologically differentiated as they ever were. A Chinaman is a Chinaman today, and as different from anybody else in the world, as he ever was. Contrast this with real biological elimination with which this Darwinian School of militaristic philosophy draws so false an analogy. What comparison exists between a Chinaman and a dinosaur? Natural selection operated with a real *Allmacht* on the dinosaurs to a finish that made literally true the proverbial statement of the wondering rustic about the giraffe: "There ain't no such animal." But the Chinaman hopelessly defeated and crushed in military affairs is still with us and quite capable of enjoying life in his peculiar way. He stands in the aggregate as a gigantic refutation of the much lauded claim which the Germans have made for the "fundamental biological basis of war."

While we are on this subject of natural selection, it will be well to examine into another aspect of the subject in its relation to war. It has been contended by various persons that war has an unfortunate selective action on the individuals engaged in it. The operation of war is supposed to be selective within the race for the elimination of the best and the preservation of the worst germ plasm. This is alleged on the general ground that the physically, mentally, and morally best of the youth of the nation are those most likely to take part in war in the first place, and in the second place, most likely, because of these characteristics, to be killed in the course of the conflict. Dire pictures have been drawn of the effect upon the race of engaging in war, through the supposed operation of this dysgenic selection. The more one examines the facts, however, the more is it apparent that the case has been very much exaggerated.

Many considerations lead to this conclusion. In the first place, the future of the race, in the narrowly biological sense, is solely dependent upon the continuity of its germ plasm. In the human species the germ plasm

of the race is equally borne by both the males and the females. But, putting the very worst complexion on the dysgenic argument, the females of the race are not eliminated in war. So that if we were to grant for the moment the contention that the best males of the race are killed off, it would still remain the fact that but very slight deleterious racial effects would result, because there would be left behind in the surviving females at least half of the total racial germ cells of all qualities. Mendel's principles of inheritance teach us that even in such an extremely unlikely circumstance that all the germ plasm borne in spermatozoa was at the end of the war of an inferior quality, it would still be possible through the operation of segregation to have again a preponderant stock of superior individuals after a few generations, provided there were no social restrictions on assortative mating, which, broadly speaking, there are not.

Furthermore, the hypothesis of racial degeneration by elimination of the best tacitly assumes that those males eliminated in battle have not left progeny before their elimination, whereas, as a matter of statistical fact, a considerable portion of them do leave behind such progeny. Again it must not be forgotten that the whole of the population, both male and female, under about twenty years of age is left untouched by war, and available for the perpetuation of the race as they grow older. This means in statistical terms, that about 40 per cent of the total male population existent at any given moment, and in which all qualities of germ plasm, good, bad, and indifferent, are normally distributed, as in a random sample of the whole, are not even involved in war and hence stand no chance to be eliminated by its operation.

In the second place, even in the most destructive of modern wars the proportion of totally eliminated casualties to the whole population is not very great. Indeed, it is always found to be surprisingly small when reviewed dispassionately by the vital statistician after the war is over. To take the case of our own Civil War, the proportion of casualties to the total population was only 2 per cent, and even in proportion to the male population within the likely breeding period (say fifteen to fifty years of age) was slightly under 9 per cent. It is, of course, too early to obtain similar estimates for the present conflict.

In the case of the present war, there are still other considerations which make it clear that any putative, deleterious, selective effect of war on the races concerned will be insignificantly slight. In all of the nations involved the fighting men have been taken practically at random from the whole population so far as germinal variations are concerned. The sound biological principle of conscription operates to leave the distribution curve of

germ-plasmic qualities essentially the same after the fighting men have been taken out as it was before. The high development of the mechanical aspects of the present war operates to the same end. Hand to hand conflicts, man against man, in direct physical struggle, are a relatively small part of the present as compared with earlier wars. The agents of destruction chiefly relied on in the present conflict are entirely impersonal and distribute their effects very largely at random. The whole mode of conduct of the present war operates to make the chances for elimination of the man carrying about within his soma the best germ plasm of the race, not substantially greater than the chances of the individual bearing the poorest germ plasm.

CONCLUSION

Except for lack of time one might go on and consider other essentially biological problems of war. We have not discussed at all those fascinatingly interesting and important problems connected with the individual's part in the actual conduct of war. A nation which would systematically and thoroughly investigate such matters as what sorts of men, physically, psychologically, and morally, make the best fighters; what biological conditions, including internal states, environmental conditions in and behind the lines, conduce to most efficient fighting; how fighters should be fed to obtain the best results; and other like problems, would be in an extremely superior position in any conflict with a group not possessed of definite scientific information on these points. At present our information regarding such matters is very largely empirical.

In conclusion, the thought I most wish to leave, and which I hope I have sufficiently elaborated and illustrated, is that while war is a biological business, to the problems of which the trained biologist could contribute much, it is *not* an absolute biological necessity. Nations neither lose nor gain biologically by war. But this does not mean that wars must not and will not be fought. As a biologist I can come to no other conclusion than that wars will occur in the future as they have in the past until such time as civilized man has become a different kind of animal than he now is. Happily every war advances him by some degree on the road to that much-to-be-desired goal.

CHAPTER XXIII

The Stability of England's Population Growth[1]

The term "vital index" has been suggested[2] as a convenient designation of the function

$$V = \frac{100\ Births}{Deaths}$$

which measures more effectively than any other demographic function yet devised the essential biological fitness of a population, in the sense of organic evolution. For a population V is the direct measure of survival value. If $V > 100$ the population is not only surviving but growing, whereas if $V < 100$ the population is starting on the road to elimination, and must eventually disappear unless the vital index gets above 100. A detailed presentation of the facts regarding the vital index of certain elements of the American population has been given in earlier chapters.[3]

In examining the effect of the Great War upon the growth of population one cannot fail to be impressed by the fact that the return of the birth/death ratio (vital index) to a normal or even super-normal value after the marked dislocation produced by the war, and at its end the superimposed influenza pandemic, was extraordinarily rapid. Indeed Pearl[4] was led to remark, after examining the matter in the chief belligerent countries, that

> Altogether, these examples, which include the effects of the most destructive war known to modern man, and the most devastating epidemic since the Middle Ages, furnish a substantial demonstration of the fact that population growth is a highly self-regulated biological phenomenon. Those persons who see in war and pestilence any absolute solution of the world problem of population, as postulated by Malthus, are optimists indeed. As a matter of fact, all history definitely tells us, and recent history fairly shouts in its

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[1] This chapter is based on a paper by Raymond Pearl and Magdalen H. Burger entitled "The vital index of the population of England and Wales, 1838–1920," published in the *Proc. Nat. Acad. Sci.*, vol. 8, pp. 71–76, 1922; and a paper by R. Pearl entitled, "Seasonal fluctuations of the vital index of a population," *ibid.*, pp. 76–78, 1922.

[2] Pearl, R., The effect of the war on the chief factors of population change, *Science* (N.S.), vol. 51, 1920, pp. 553–556.

[3] Pearl, The vitality of the peoples of America, *Amer. J. Hyg.*, vol. 1, 1921, pp. 592–674. See Chapters VIII and IX, *supra*.

[4] Pearl, A further note on war and population, *Science* (N.S.), vol. 53, 1921, pp. 120-121.

emphasis, that such events make the merest ephemeral flicker in the steady onward march of population growth.

In view of the fact that this function V has so quickly returned to normal, or approximately normal, values after the recent disturbances it is a matter of considerable interest and significance to the student of problems of public health in the broad sense, of population growth, and of human ecology, to see how precisely the self-regulatory mechanism of populations operates over long periods of time. To this end we have examined all the existing continuous statistics of England and Wales.

Table 166 gives the vital index (100 births/deaths) for the population of England and Wales, in each quarter of the years 1838 to 1920 inclusive. The data from which the computations were made were taken from official sources.[5]

The immediately striking feature of this table is the relative constancy of the values as one runs down the columns. In order to appreciate this fact fully, however, it is necessary to resort to graphical presentation. As the data of table 166 are too detailed to make such an examination of the trend feasible within the space of a single printed page, the data have been combined in quinquennial periods in table 167. In this table are given, for each five-year period, the total births, total deaths, vital index, and crude birth rate. The latter figures have been calculated directly from the birth and population figures and differ slightly (but in no wise significantly) from the crude birth rates in Annual Reports of the Registrar-General, chiefly because of the fact that we have used a different quinquennial grouping.

The vital index and birth rate data from table 167 are shown graphically in figure 97.

The diagram is plotted on an arithlog grid, in order that the slopes of the lines may be strictly comparable.[6]

It is at once apparent that *the ratio of births to deaths in England and Wales had a slow but extremely even and steady increase from 1838 to 1914.* This steady progress was interrupted to a degree sufficient to be apparent upon only two occasions during the three quarters of a century. These were

[5] *Quarterly Return of Marriages, Births, and Deaths Registered in England and Wales, etc.* Published by Authority of the Registrar-General. London. H. M. Stationery Office. *Passim.* Also corresponding Weekly Return, and Annual Reports.

[6] For a discussion of the significance and advantages of plotting trend lines on a logarithmic scale, *cf.* Fisher, I., The "ratio" chart for plotting statistics, *Q. P. Am. Stat. Ass.*, vol. 15, 1917, pp. 577–601, and Field, J. A., Some advantages of the logarithmic scale in statistical diagrams, *J. Pol. Econ.*, vol. 25, 1917, pp. 805–841.

in 1847 to 1849 and 1890 to 1892. These fluctuations, which only slightly affected the even upward trend of the curve were due to the influenza pandemics of 1847 to 1848 and 1890 to 1891. The broad result is perfectly

TABLE 166

The number of births to each 100 deaths in England and Wales in each quarter of the years 1838 to 1920, inclusive

QUARTERS ENDED					QUARTERS ENDED					QUARTERS ENDED				
Year	March	June	September	December	Year	March	June	September	December	Year	March	June	September	December
1838	116	134	157	140	1866	142	150	153	158	1894	155	186	201	180
1839	138	146	157	141	1867	145	178	176	158	1895	141	179	174	161
1840	134	143	148	135	1868	166	184	147	160	1896	163	185	181	168
1841	135	151	164	150	1869	153	159	166	149	1897	164	179	169	170
1842	141	155	150	148	1870	143	168	155	151	1898	154	183	166	169
1843	144	150	167	150	1871	151	167	159	144	1899	157	182	151	152
1844	142	160	163	143	1872	155	173	170	176	1900	132	166	175	167
1845	137	153	177	163	1873	163	174	178	160	1901	158	182	169	167
1846	162	166	136	128	1874	157	176	169	150	1902	153	179	209	168
1847	122	130	136	123	1875	132	164	174	159	1903	170	195	207	169
1848	116	150	160	144	1876	162	179	180	177	1904	157	192	178	165
1849	145	150	100	139	1877	171	170	195	177	1905	160	192	195	172
1850	147	168	171	159	1878	158	177	172	155	1906	169	189	181	166
1851	149	160	165	150	1879	145	167	210	160	1907	143	187	216	161
1852	152	158	151	152	1880	152	186	167	165	1908	153	201	208	170
1853	137	147	160	140	1881	163	186	196	177	1909	143	189	218	172
1854	144	168	136	133	1882	159	182	186	163	1910	163	203	218	168
1855	123	155	177	153	1883	158	171	189	167	1911	156	188	158	169
1856	164	173	173	164	1884	170	181	167	165	1912	154	188	216	170
1857	157	170	160	146	1885	157	168	191	173	1913	148	187	199	172
1858	136	158	160	133	1886	147	184	179	168	1914	149	182	197	159
1859	144	166	161	155	1887	153	176	178	163	1915	125	154	180	134
1860	149	157	190	158	1888	149	179	199	169	1916	136	165	193	134
1861	143	172	170	159	1889	158	183	180	165	1917	109	136	172	136
1862	149	173	187	150	1890	136	171	180	139	1918	118	144	147	67
1863	145	160	154	155	1891	145	139	192	156	1919	75	137	197	195
1864	135	161	161	144	1892	120	176	197	168	1920	197	208	245	180
1865	138	167	160	148	1893	160	177	160	147					

clear and outstanding. The population of England and Wales is today biologically fitter and possessed of greater purely biological survival value as a whole population than it was three quarters of a century ago. Whether it is a mentally, morally or anthropometrically fitter population does not

now concern us. We are dealing at the moment solely with the fact that, taking the people of England and Wales as a whole, slightly over 2 babies were born for every death in a year in 1920, as against 1.4 babies per death in a year in 1838 to 1839.

Now this result, which is beyond any question a fact, will strike anyone informed as to the sociological and eugenical literature of the last two decades as curiously at variance with the pessimistic tenor of that literature, taken as a whole. It has been pronounced from high places that the general trend of the British people was biologically downwards, that they

TABLE 167

Grouped data for vital index and crude birth rate

PERIOD	BIRTHS	DEATHS	100 BIRTHS / DEATHS	GENERAL BIRTH RATE PER 1000 POPULATION
1838–1839	956,361	681,744	140.28	31.0
1840–1844	2,600,288	1,756,431	148.04	32.2
1845–1849	2,797,329	2,003,657	139.61	32.6
1850–1854	3,080,095	2,030,528	151.69	33.9
1855–1859	3,300,929	2,126,461	155.23	34.3
1860–1864	3,560,830	2,263,769	157.30	34.9
1865–1869	3,830,527	2,438,121	157.11	35.3
1870–1874	4,100,856	2,541,625	161.35	35.5
1875–1879	4,399,070	2,623,391	167.69	35.6
1880–1884	4,451,771	2,591,038	171.81	33.8
1885–1889	4,450,173	2,620,108	169.85	32.0
1890–1894	4,486,912	2,778,642	161.48	30.5
1895–1899	4,611,116	2,771,151	166.40	29.6
1900–1904	4,691,038	2,739,365	171.25	28.5
1905–1909	4,637,271	2,613,992	177.40	26.7
1910–1914	4,411,823	2,519,713	175.09	24.2
1915–1919	3,623,894	2,685,456	134.95	20.8
1920	957,994	466,213	205.48	25.5

were in fact becoming biologically dangerously near to a decadent race. Abundant quotations in support of this contention could be cited, were space available and were it necessary. This gloomy view has had its foundation solely upon the fact that, since the quinquennium 1875 to 1880, the birth rate in England and Wales has been falling rather rapidly, as is clearly shown in figure 97. This fact has been brought out by Elderton[7] in great detail.

[7] Elderton, E. M., Report on the English birth rate. Part I. England, north of the Humber, *Eugenics Lab. Mem.*, 19 and 20, Cambridge Univ. Press, 1914, pp. viii and 246.

But from a purely biological view-point, what matters a falling birth rate if the death rate falls even more rapidly, so that the net survivorship at any instant of time is constantly getting higher? To this it will, of course, be answered at once by those who view with alarm the declining birth rate that the real crux of the matter is in the differential change in fertility. Nowadays the "best" people do not produce their due share of progeny, while the "worst" people over-produce. If one accepts the man-made definitions of "best" and "worst" this is plainly true for some

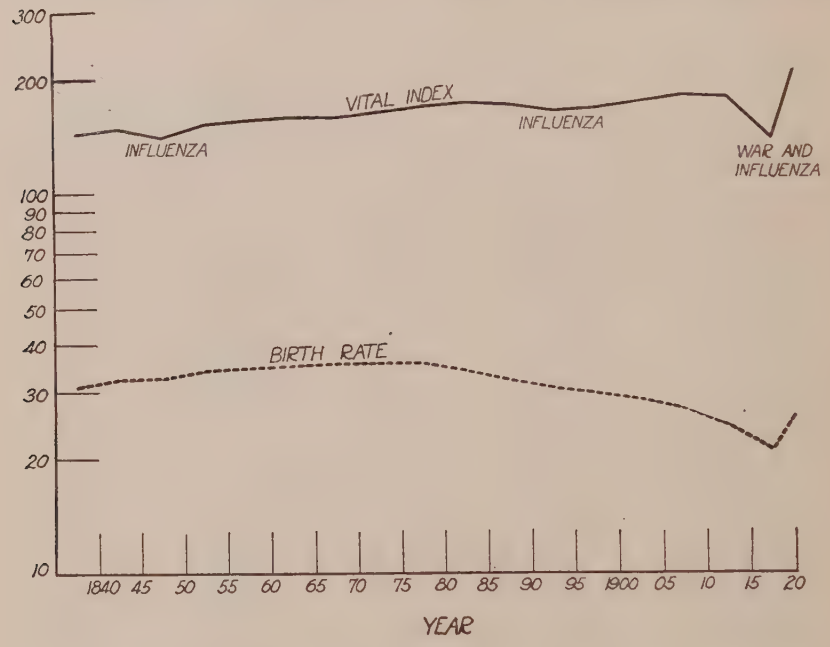

Fig. 97. Trend of Vital Index (100 Births/Deaths) and Crude Birth Rate in England and Wales, 1838 to 1920, Inclusive

civilizations at least. In the American population, however, as Pearl has recently shown, the element probably least desirable racially, the negro, has the lowest survival value as a group (vital index generally less than 100). But is old Dame Nature ever really interested in any test of "best" and "worst" *except survival?* There is no evidence as yet that she is. Measured by this test the population as a whole of England and Wales is today biologically more vigorous than it was in 1838. This is a plain *fact.* Whether this fact is, sociologically or eugenically considered, a bad

thing or a good thing, is something more than a purely biological problem, and as such we are content to leave it to the discussion of others. What seems to us of considerable interest biologically, is the extraordinary perfection, demonstrated in the figures here presented, of the self-regulation of population growth. Disturbances of the course of the birth rate have been compensated for, in the population and period under review, with the greatest nicety.

SEASONAL FLUCTUATIONS OF THE VITAL INDEX

Table 166 gives the necessary data for finding, for one population, that of England and Wales, the course of the seasonal fluctuation of this important demographic constant.

Table 168 gives the frequency distributions for variation in the value of the vital index of the population of England and Wales in each of the four quarters of the year, ending, respectively, on March 31, June 30, September 30, and December 31, during the years 1838 to 1920, inclusive.

The significant biometric constants from table 168 are presented in table 169. It is apparent that there are a few highly aberrant observations in the series, but it has not been thought best to discard or adjust them in this first treatment of the subject. Consequently it is to be understood that all the values given in table 168 were used in computing the values in table 169.

From these data the following points are to be noted:

1. The vital index has the lowest mean value in the quarter ending on March 31, the winter quarter. In that period the birth incidence is relatively low and the death incidence relatively high.

2. Next in value to this, but standing 9.40 ± 1.75 points above it, is the mean vital index for the autumn quarter ending December 31. The difference being 5.4 times its probable error, may be regarded as significant.

3. The spring quarter, ending June 30, shows the next higher mean, being 13.43 ± 1.75 points above the winter quarter.

4. The highest value of the index falls in the summer quarter, when births are most frequent and deaths least so. The mean value, however, lies only 5.54 ± 2.05 above that for the spring quarter, a difference which cannot be regarded as significant.

5. In variability of the vital index, the first two and the last quarters of the year, all exhibit significantly the same status. The vital index is distinctively more variable in the summer quarter, the difference in standard deviations when this quarter is compared with that ending June 30, amounting to 6.04 ± 1.45. This may be regarded as significant, being 4.2 times its probable error.

TABLE 168

Frequency distributions for variation in the vital index in the four quarters of the year

VITAL INDEX	QUARTERS ENDING			
	March 31	June 30	September 30	December 31
65– 69				1
70– 74				
75– 79	1			
80– 84				
85– 89				
90– 94				
95– 99				
100–104			1	
105–109	1			
110–114				
115–119	3			
120–124	3			1
125–129	1			1
130–134	3	2		4
135–139	9	3	3	4
140–144	11	2		7
145–149	13	2	3	5
150–154	9	7	5	9
155–159	13	6	5	11
160–164	10	5	10	9
165–169	2	11	9	17
170–174	3	9	8	7
175–179		11	8	4
180–184		9	6	2
185–189		10	3	
190–194		2	4	
195–199	1	1	8	1
200–204		2	1	
205–209		1	3	
210–214			1	
215–219			4	
245–249			1	
Total	83	83	83	83

TABLE 169

Biometric constants deduced from table 168

QUARTER ENDING	MEAN	STANDARD DEVIATION
March 31	147.06 ± 1.21	16.32 ± 0.85
June 30	169.89 ± 1.21	16.35 ± 0.86
September 30	175.43 ± 1.66	22.39 ± 1.17
December 31	156.46 ± 1.27	17.14 ± 0.90

It thus appears that the extremely close compensatory relation between birth rate and death rate, which has been shown to hold in annual figures, does not obtain within the single year. Instead there is a well-marked statistically significant intra-annual, or seasonal fluctuation of the birth-death ratio.

CHAPTER XXIV

The Curve of Population Growth[1]

It is obviously possible in any country or community of reasonable size to determine an empirical equation, by ordinary methods of curve fitting, which will describe the normal rate of population growth. Such a determination will not necessarily give any inkling whatever as to the underlying organic laws of population growth in a particular community. It will simply give a rather exact empirical statement of the nature of the changes which have occurred in the past. No process of empirically graduating raw data with a curve can in and of itself demonstrate the fundamental causes which underlie the occurring change. It can at best have but inferential significance; it can never be of demonstrative worth. To suppose that it can is a process of reasoning which assumes a fundamental or necessary relationship to exist between two sets of phenomena because the same curve describes the quantitative relations of both sets. A little consideration indicates that this method of reasoning certainly can not be of general application, even though we assume it to be correct in particular cases. The difficulty arises from the fact that the mathematical functions commonly used with adequate results in physical, chemical, biological and mathematical investigations are comparatively few in number. The literature of science shows nothing clearer than that the same type of curve frequently serves to describe with complete accuracy the quantitative relations of widely different natural phenomena. As a consequence any proposition to conclude that two sets of phenomena are causally or in any other way fundamentally related solely because they are described by the same type of curve is of very doubtful validity. A few examples will make clear the point here under discussion.

Armsby[2] showed that the rate of gain of protein per thousand pounds live weight in growing animals follows extremely closely the following curve:

[1] This chapter presents, with considerable additions, the substance of three papers by Raymond Pearl and Lowell J. Reed, On the rate of growth of the population of the United States since 1790 and its mathematical representation, *Proc. Nat. Acad. Sci.*, vol. 6, pp. 275–288, 1920; On the mathematical theory of population growth, *Metron*, vol. 3, pp. 6–19, 1923; and The probable error of certain constants of the population growth curve, *Amer. Jour. Hygiene*, May, 1924.

[2] Armsby, H. P., Feeding for meat production, *Bureau of Animal Industry, Bulletin 108*, pp. 1–89, 1908.

$g = 135/(a + 20)$, where g is gain in protein per day per 1000 pounds live weight and a is age in days. This curve, as his figure 1 clearly shows, fits the observational data at hand remarkably well. This equation is the equation of a rectangular hyperbola. But it is a well-known fact that the relation between degree of dissociation and degree of dilution in dilute solutions is given by a hyperbola. Now in so far there would appear to be exactly the same kind of logical basis for the conclusion that since the same curve describes the rate of protein gain in growing animals as describes dissociation phenomena, therefore rate of protein gain is a dissociation phenomenon, as would exist for the conclusion that growth is an autocatalytic reaction provided there were good agreement between observed and theoretical curves in the latter case.[3] But the most ardent advocate of the plan of deducing fundamental relationships from similarity of curve type would not maintain that the rate of protein gain in growing animals is in any causal or fundamental way directly related to the phenomenon of dissociation in dilute solutions.

Let us take another case. One of the fundamental gas laws is that the "pressure of any given mass of gas varies directly as the absolute temperature if the volume of the gas remains constant." The mathematical expression of this relation is the equation of a straight line. Now Galton, Pearson and their co-workers have shown, with a wealth of data drawn from man and other organisms, that the regression of offspring on parent in parental inheritance is a linear function. If the mean conditions of a characteristic of the offspring of each group of parents be plotted these plotted points will fall on a straight line, within the errors of random sampling. This result rests on a great mass of exact measurements. But of course no one would attempt seriously to maintain that parental inheritance and regression are phenomena of gas pressure.

The point is this: If there is good evidence *on other than quantitative grounds* that two sets of phenomena are qualitatively alike it is pertinent and significant to present as additional and confirmatory evidence data tending to show that these sets of phenomena are similar in their quantitative relations. But similarity of quantitative relations between phenomena can not safely be taken as proof (or, in the absence of qualitative data sufficient alone practically to establish the point, even as particularly

[3] *Cf.* Robertson, T. B., On the normal rate of growth of an individual and its biochemical significance, *Arch. f. Entwicklungsmech.*, Bd. 25, pp. 581–614, 1908. Further remarks on the normal rate of growth of an individual and its biochemical significance, *ibid.*, Bd. 26, pp. 108–118, 1908. There have also been many later papers by the same author on this subject, summarized in his book.

weighty evidence) of qualitative identity, because of the observed general lack of uniqueness in the quantitative relations of natural phenomena. In a word the final *proof* of qualitative identity of phenomena must always in last analysis be qualitative in its nature; quantitative evidence in such cases can at best have but an inferential confirmatory bearing on the qualitative point at issue.

In spite of the fact that most mathematical expressions of population growth are purely empirical, they have a distinct and considerable usefulness. This usefulness arises out of the fact that actual counts of population by census methods are made at only relatively infrequent intervals, usually ten years and practically never oftener than five years. For many statistical purposes, it is necessary to have as accurate an estimate as possible of the population in inter-censal years. This applies not only to the years following that on which the last census was taken, but also to the intercensal years lying between prior censuses. For purposes of practical statistics it is highly important to have these inter-censal estimates of population as accurate as possible, particularly for the use of the vital statistician, who must have these figures for the calculation of annual death rates, birth rates, and the like.

The usual method followed by census offices in determining the population in inter-censal years is of one or the other of two sorts, namely, by arithmetic progression or geometric progression. These methods assume that for any given short period of time the population is increasing either in arithmetic or geometric ratio. Neither of these assumptions is ever absolutely accurate even for short intervals of time, and both are grossly inaccurate for the United States, at least, for any considerable period of time. What actually happens is that following any census estimates are made by one or another of these methods of the population for each year up to the next census, on the basis of data given by the last two censuses only. When that next census has been made, the previous estimates of the inter-censal years are corrected and adjusted on the basis of the facts brought out at that census period.

Obviously the best general method of estimating population in inter-censal years is that of fitting an appropriate curve to *all* the available data, and extrapolating for years beyond the last census, and reading off from the curve values for inter-censal years falling between earlier censuses. The methods of arithmetic or geometric progression use only two census counts at the most. Fitting a curve to all the known data regarding population by the method of least squares must obviously give a much sounder and more accurate result. In making this statement, one realizes perfectly,

of course, the dangers of extrapolation. These dangers have been well emphasized by Perrin,[4] who used higher order parabolas to predict the future population of Buenos Aires. In keeping sharply before our minds the dangers of extrapolation from a curve, we are apt to forget that the methods of extrapolation by arithmetic or geometric progression have much less general validity than from a curve, and the inaccuracies are found in practice, except by the rarest of accidents, to be actually greater.

The first one to attempt an adequate mathematical representation of the normal rate of growth of the population of the United States was Pritchett.[5] Taking the census data from 1790 to 1880, inclusive, Pritchett fitted by the method of least squares the following equation:

$$P = A + Bt + Ct^2 + Dt^3 \qquad \text{(i)}$$

where P represents the population and t the time from some assumed epoch. As a matter of fact, Pritchett took the origin of the curve at 1840, practically the center of the series. With this third-order parabola Pritchett got a very accurate representation of the population between the dates covered. As will presently appear this curve did not give, even within the period covered, as accurate results as a more adequate curve would have done, and it overestimated the population after a very short interval beyond the last observed ordinate as is shown in table 171.

Some seventeen years ago Pearl[6] demonstrated the applicability of a logarithmic curve of the form

$$y = a + bx + cx^2 + d \log x \qquad \text{(ii)}$$

to the representation of growth changes, using the aquatic plant *Ceratophyllum* as material. Following the application of this curve to growth of this plant it was found equally useful in representing a wide range of other growth and related changes.[7] This list now includes, of matters

[4] Perrin, E., On some dangers of extrapolation, *Biometrika*, vol. 3, 1904, pp. 99–103.

[5] Pritchett, A. S., A formula for predicting the population of the United States, *Quart. Publ. Amer. Statistical Assoc.*, vol. 2, 1891, pp. 278–286.

[6] Pearl, R., Variation and differentiation in ceratophyllum, *Carnegie Inst. Washington, Publ.*, vol. 58, 1907 (136).

[7] *Cf.* the following papers:

Curtis, M. R., 1914, A biometrical study of egg production in the domestic fowl. IV. Factors influencing the size, shape, and physical constitution of eggs, *Arch. Entwicklungsmech. Organ.*, B. 39, Heft 2/3, pp. 217–327.

Pearl, R., 1909, Studies on the physiology of reproduction in the domestic fowl. I. Regulation in the morphogenetic activity of the oviduct, *J. Exp. Zool.*, vol. 6, no. 3, pp. 339–359.

Pearl, R., 1914, On the law relating milk flow to age in dairy cattle, *Proc. Soc. Exp. Biol. Med.*, vol. 12, no. 1, pp. 18–19.

worked out in the Biological Laboratory of the Maine Experiment Station, such diverse phenomena as change of size of egg with successive layings, change of milk production with age, etc. Donaldson and Hatai[8] have demonstrated the applicability of this type of equation to bodily growth in the white rat and frog.

While the increase in size of a population cannot on *a priori* grounds be regarded, except by rather loose analogy, as the same thing as the growth of an organism in size, nevertheless it is essentially a growth phenomenon. It, therefore, seems entirely reasonable that this type of curve should give a more adequate representation of population increase than a simple third-order parabola. The actual event justifies this assumption, as will presently appear.

Table 170 shows the counted population as determined by the Census Bureau on the dates mentioned from 1790 to 1910. The exact dates were furnished in a personal communication from the present Director of the Census. These figures embody some adjustments and corrections made by the Census Bureau since the original censuses were made.

To the data of table 170 the following equation was fitted by the method of least squares, taking origin at 1780, and making due allowance in the abscissal intervals for the actual dates of the several censuses:

$$y = a + bx + cx^2 + d \log x$$

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[8] Donaldson, H. H., 1908, A comparison of the albino rat with man in respect to the growth of the brain and of the spinal cord, *J. Compar. Neurol. Psych.*, vol. 18, no. 4, pp. 345–389.

Donaldson, H. H., 1909, On the relation of the body length to the body weight and to the weight of the brain and of the spinal cord in the albino rat (*Mus norvegicus* var. *albus*), *ibid.*, vol. 19, no. 2, pp. 155–167.

Donaldson, H. H., 1910, On the percentage of water in the brain and in the spinal cord of the albino rat, *ibid.*, vol. 20, no. 3, pp. 119–144.

Donaldson, H. H., 1911, On the regular seasonal changes in the relative weight of the central nervous system of the leopard frog, *J. Morph.*, vol. 22, pp. 663–694.

Donaldson and Hatai, Shinkishi, 1911, A comparison of the Norway rat with the albino rat in respect to body length, brain weight, spinal cord weight, and the percentage of water in both the brain and the spinal cord, *J. Compar. Neurol. Psych.*, vol. 21, pp. 417–458.

Hatai, Shinkishi, 1909, Note on the formulas used for calculating the weight of the brain in the albino rats, *ibid.*, vol. 19, no. 2, pp. 169–173.

Hatai, Shinkishi, 1911, A formula for determining the total length of the leopard frog (*R. pipiens*) for a given body weight, *Anat. Rec.*, vol. 5, no. 6, pp. 309–312.

Hatai, Shinkishi, 1911, An interpretation of growth curves from a dynamical standpoint, *ibid.*, vol. 5, no. 8, pp. 373–382.

TABLE 170

Showing the dates of the taking of the census and the recorded populations from 1790 to 1910

DATE OF CENSUS		RECORDED POPULATION (REVISED FIGURES FROM STATISTICAL ABSTRACT, 1918)
Year	Month and day	
1790	First Monday in August	3,929,214
1800	First Monday in August	5,308,483
1810	First Monday in August	7,239,881
1820	First Monday in August	9,638,453
1830	June 1	12,866,020
1840	June 1	17,069,453
1850	June 1	23,191,876
1860	June 1	31,443,321
1870	June 1	38,558,371
1880	June 1	50,155,783
1890	June 1	62,947,714
1900	June 1	75,994,575
1910	April 15	91,972,266

TABLE 171

Showing (a) the actual population on census dates, (b) estimated population from Pritchett's third-order parabola, (c) estimated population from logarithmic parabola, and (d) (e) root-mean square errors of both methods*

CENSUS YEAR	(a) OBSERVED POPULATION	(b) PRITCHETT ESTIMATE	(c) LOGARITHMIC PARABOLA ESTIMATE	(d) ERROR OF (b)	(e) ERROR OF (c)
1790	3,929,000	4,012,000	3,693,000	+ 83,000	− 236,000
1800	5,308,000	5,267,000	5,865,000	− 41,000	+ 557,000
1810	7,240,000	7,059,000	7,293,000	− 181,000	+ 53,000
1820	9,638,000	9,571,000	9,404,000	− 67,000	− 234,000
1830	12,866,000	12,985,000	12,577,000	+ 119,000	− 289,000
1840	17,069,000	17,484,000	17,132,000	+ 415,000	+ 63,000
1850	23,192,000	23,250,000	23,129,000	+ 58,000	− 63,000
1860	31,443,000	30,465,000	30,633,000	− 978,000	− 810,000
1870	38,558,000	39,313,000	39,687,000	+ 755,000	+1,129,000
1880	50,156,000	49,975,000	50,318,000	− 181,000	+ 162,000
1890	62,948,000	62,634,000	62,547,000	− 314,000	− 401,000
1900	75,995,000	77,472,000	76,389,000	+1,477,000	+ 394,000
1910	91,972,000	94,673,000	91,647,000	+2,701,000	− 325,000
				935,000†	472,000†
1920		114,416,000	108,214,000		

* To the nearest thousand.

† Root-mean square error.

where y denotes population and x time. The actual equation deduced was

$$y = 9{,}064{,}900 - 6{,}281{,}430x + 842{,}377x^2 + 19{,}829{,}500 \log x \qquad \text{(iii)}$$

The results are set forth in table 171, where Pritchett's figures are given for comparison.

It is obvious from the data of table 171 that, with the same number of constants, the logarithmic parabola gives a distinctly better graduation than a third-order parabola.

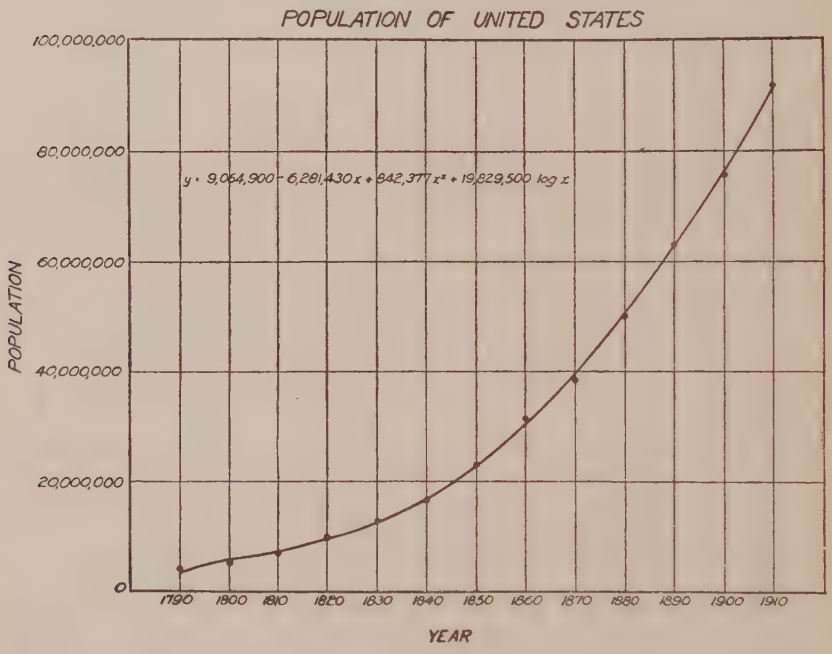

FIG. 98. DIAGRAM SHOWING OBSERVED AND CALCULATED POPULATIONS (FROM LOGARITHMIC PARABOLA) FROM 1790 TO 1920

The extreme precision of the present graduation is shown graphically in figure 98.

It is evident that as a purely empirical representation of population growth in the United States equation (iii) gives results of a very high degree of accuracy. Indeed, interpolation on this curve for inter-censal years may obviously be relied upon with a greater probability that the estimated figures approximate the unknown true facts than is afforded by any other estimating expedient hitherto applied to the known data.

An indication of the general exactness of this curve (iii) for estimating future population by extrapolation may be got in the following way. Suppose a mathematician of the Civil War period had desired to estimate the population of the United States in 1910, and had fitted a curve of the type of (ii), by the method of least squares to the known data available to him, namely, the census counts of 1790 to 1860, inclusive, he would have got this result:

$$y = 8{,}619{,}800 - 5{,}680{,}540x + 822{,}709x^2 + 16{,}987{,}200 \log x \qquad \text{(iv)}$$

If he had calculated from this equation the probable population in 1910, the figure he would have obtained would have been 92,523,000, a result only approximately a half million, or 0.6 per cent in error, as subsequent events proved. A prophecy less than 1 per cent in error of an event to happen fifty years later is undeniably good predicting.

It is of interest to exhibit the equations and results in predicting the 1910 population obtained by fitting our logarithmic parabola to the data available after the completion of each successive census from 1870 on. We have:

Data for 1790 to 1870, inclusive:

$$y = 8{,}287{,}700 - 5{,}300{,}270x + 795{,}540x^2 + 15{,}778{,}000 \log x \qquad \text{(v)}$$

Predicted population in 1910 = 91,201,000
Deviation of prediction from actual, 1910 = −771,000
Percentage error = 0.8 per cent
Data for 1790 to 1880, inclusive:

$$y = 7{,}981{,}100 - 4{,}971{,}040x + 764{,}896x^2 + 14{,}993{,}500 \log x \qquad \text{(vi)}$$

Predicted population in 1910 = 89,128,000
Deviation of prediction from actual, 1910 = −2,844,000
Percentage error = 3 per cent
Data for 1790 to 1890, inclusive:

$$y = 9{,}013{,}800 - 6{,}242{,}170x + 839{,}782x^2 + 19{,}744{,}300 \log x \qquad \text{(vii)}$$

Predicted population in 1910 = 91,573,000
Deviation of prediction from actual, 1910 = −399,000
Percentage error = 0.4 per cent
Data for 1790 to 1900, inclusive:

$$y = 8{,}748{,}000 - 5{,}880{,}890x + 821{,}001x^2 + 18{,}232{,}100 \log x \qquad \text{(viii)}$$

Predicted population in 1910 = 91,148,000
Deviation of prediction from actual, 1910 = −824,000
Percentage error = 0.9 per cent

Beginning with 1860 (equation (iv)) and coming down to 1900, our hypothetical statistician would have been only once in error as much as 1 per cent in his prediction of the 1910 population by this logarithmic parabola. The one larger error is for the 1880 curve, where apparently the aberrant counts of 1860 and 1870 exert an undue influence.

Altogether it seems justifiable to conclude that:

1. A logarithmic parabola of the type of equation (ii) describes the changes which have occurred in the population of the United States in respect of its gross magnitude, with a higher degree of accuracy than any empirical formula hitherto applied to the purpose.

2. The accuracy of the graduation given by this logarithmic parabola is entirely sufficient for all practical statistical purposes.

Satisfactory as the empirical equation above considered is from a practical point of view, it remains the fact that it is an empirical expression solely, and states no general law of population growth. Insofar it is obviously an undesirable point at which to leave the problem of the mathematical expression of the change of population in magnitude.

It is quite clear on *a priori* grounds, as was first pointed out by Malthus in non-mathematical terms, that in any restricted area, such as the United States, a time must eventually come when population will press so closely upon subsistence that its rate of increase per unit of time must be reduced to the vanishing point. In other words, a population curve may start, as does that shown in figure 98, with a convex face to the base, but presently it must develop a point of inflection, and from that point on present a concave face to the x axis, and finally become asymptotic, the asymptote representing the maximum number of people which can be supported on the given fixed area.[9] Now, while an equation like (ii) can, and will in due time, develop a point of inflection and become concave to the base it never can become asymptotic. It, therefore, cannot be regarded as a hopeful line of approach to a true law of population growth.

What we want obviously is a mathematical picture of the *whole* course of population in this country. It is not enough to be able to predict twenty or fifty years ahead as our logarithmic parabola is able to do satisfactorily, *in one portion of the whole curve*. How absurd equation (iii) would be over a really long time range is shown if we attempt to calculate from it the probable population in, say, 3000 A.D. It gives a value of 11,822,000,000.

[9] Always, be it clearly understood, on the assumption that the average standard of living, method of agricultural production, etc., either do not change at all in the period between the end of historical past record of fact and the time when dy/dx becomes negligibly small as the asymptote is approached, or that the net effective magnitude of any such changes as do occur will be relatively so small as to be negligible in comparison with the effect of such factors as reproduction and immigration in determining the relation between population and time in an area of fixed limits. In any mathematical treatment of the subject these factors of standard of living, methods of agriculture, etc., represent essentially constant (and hence omitted) parameters of any assumed functional relation between population and time within a given epoch or cycle.

But this is manifestly ridiculous; it would mean a population density of 6.2 persons per acre or 3968 persons per square mile.

It would be the height of presumption to attempt to predict *accurately* the population a thousand years hence. But any real law of population growth ought to give some general and approximate indication of the number of people who would be living at that time within the present area of the United States, provided no cataclysmic alteration of circumstances has in the meantime intervened.

MATHEMATICAL THEORY

Careful study of the matter will convince one that at least the factors listed below must be taken account of in any mathematical theory of population growth which aims at completeness. The necessity for a part of these factors is evident on purely *a priori* grounds. The remainder are equally obvious and certain deductions from observed facts as to how populations do actually grow.

1. If any discussion of the growth of human population is to be profitable in any real or practical sense, the *area* upon which the population grows must be taken as a finite one with definite limits, however large. For the growth of human populations the upper limit of finite areas possible of consideration, must plainly be the habitable area of the earth. Smaller areas, as politically defined countries, may be treated each by itself. But whether this is done or not, there clearly is an upper finite limit of area on which *human* population can grow.

2. If there is a finite upper limit to the *area* upon which population may grow, then with equal clearness there must be a finite upper limit to *population* itself, or in other words to the number of persons who can live upon that area. It is obvious for example that it is a biological impossibility for so many as 50,000 human beings to live, and derive support for living, upon one acre of ground, provided every other acre of the possibly habitable area of the earth is at the same time inhabited to the same degree of density. This is obviously true whatever the future may hold in store for us in the way of agricultural discoveries, improvements, or advancements. That there is a finite upper limit to the population which can live upon a finite area (as of the earth) is really as much a physical as a biological matter. The amount of water which can be contained in a pint measure is strictly limited to a pint. It cannot by any chance be 10 gallons.

And this conclusion is in no way determined or limited by the present limitations of our knowledge of physics. Nor can it be upset by any future discoveries to be made in the realm of physics. It is this point which is so

usually overlooked by editorial writers for newspapers, and other not particularly deep thinkers upon the problem of population. From Malthus to the present time, everyone who has pointed out that there must be some upper limit to human population upon this globe, has been met by the contention that he has overlooked the possibilities inherent in the future development of science. Of course, future scientific discoveries can have no bearing upon the bald fact that there must somewhere be an upper limit to population. They can only influence the precise location (or magnitude) of that upper limit. But the discrimination between these two ideas appears to be too much for many human minds. Mathematical theories of population even have been more or less seriously advanced which really postulated that with the passage of time the curve of population would run off to infinity! Of course attention was not drawn to this feature of such theories, but nevertheless it was inherent and implicit in them.

3. The lower limit to population is zero. Negative populations are unthinkable in any common, practical sense.

4. History tells us what common-sense indicates *a priori*, namely that each advancement in cultural level has brought with it the possibility of additional population growth within any defined area. In the hunting stage of human culture the number of persons who can be supported upon a given area is small. In the pastoral stage of culture more persons can subsist upon a given area, though the absolute number is still small. In the general agricultural stage of civilization the possibilities of population per unit of area become again enhanced. The commercial and industrial stages of culture permit great increases of population, provided, of course, (and only under this condition) that there still remain somewhere else less densely populated areas where the means of subsistence can be produced in excess of local needs. In other words, each geographical unit which has been inhabited for any long time has, so far as the evidence available indicates, had a succession of waves or eras of population growth, each superposed upon the last, and each marking the duration of a more or less definite cultural epoch.

5. Within each cultural epoch or cycle of population growth the rate of growth of population has not been constant in time. Instead the following course of events has apparently occurred generally, and indeed almost universally. At first the population grows slowly, but the rate constantly increases to a certain point where it, the *rate* of growth, reaches a maximum. This point may presumably be taken to present the optimum relation between numbers of people and the subsistence resources of the defined area. This point of maximum rate of growth is the point of inflection of the popu-

lation growth curve. After that point is passed the rate of growth becomes progressively slower, till finally the curve stretches along nearly horizontally, in close approach to the upper asymptote which belongs to the particular cultural epoch and area involved.

All of these factors must certainly be taken account of in a mathematical theory of population growth. For convenience they may be recapitulated in brief as follows:

1. Finite limit of area
2. Upper limiting asymptote of population
3. Lower limiting asymptote of population = 0
4. Epochal (cultural) or cyclical character of growth, successive cycles being additive
5. General shape of curve of growth

With these fundamental postulates in mind we may now proceed to their mathematical expression and generalization. In our first paper we[10] took as a first approximation to the law expressing normal population growth

$$y = \frac{be^{ax}}{1 + ce^{ax}} \tag{ix}$$

This satisfied perfectly postulates 1, 2, 3, and in a fair degree 5. It made no attempt to satisfy 4. After our first paper was published we learned that nearly three-quarters of a century ago, a Belgian mathematician, Verhulst,[11] in two long since forgotten papers, which appear never to have been noted generally in the later literature on population, anticipated us in the use of the equation (ix) to represent population growth. The only recent writer on the subject who seems to have known of Verhulst's work is Du Pasquier,[12] who himself makes use of a slight and, as it seems to us, entirely unjustified and in practice usually incorrect modification of (ix). There have, of course been many attempts at getting mathematical expressions of population growth, or of growth in general. We shall make no attempt to review all this literature, chiefly for the reason that most of the mathematical expressions brought forward have been wholly lacking in generality. They have been special curves, doctored up with greater or less skill, to fit a particular set of observations, often involving assumptions

[10] Pearl, R., and Reed, L. J., *loc. cit.*

[11] Verhulst, P. F., Recherches mathématiques sur la loi d'accroissement de la population, *Mem. de l'Acad. roy. de Bruxelles*, T. XVIII, pp. 1–58, 1844.

Idem. Deuxième mémoire sur la loi d'accroissement de la population, *ibid.*, T. XX, pp. 1–52, 1846.

[12] Du Pasquier, L. G., Esquisse d'une nouvelle théorie de la population, *Vierteljahrschr. der Naturforsch. Ges. Zürich*, Jahrg. 63, pp. 236–249, 1918.

which could not possibly hold in any general law of growth. A paper by Lehfeldt[13] develops an idea as to the changes of a variable in time, which fundamentally seems to be similar to that set forth in the present discussion. He says:

Let q be the quantity whose changes in time t are to be studied. It is not to be expected that the changes of q itself should be symmetrical in time, for all the changes observed in the later half of the period of change refer to values of q larger—possibly many times larger—than in the earlier half. But log q may very possibly undergo symmetrical changes, so we will assume that it is a "normal error function" of the time, *i.e.*,

$$\log q = \log q_o + kF\left(\frac{t}{T}\right)$$

where q_o is the value of q at a certain moment (the "epoch"): t is the time in years before or after the epoch: T is a constant period and

$$F(x) = \frac{1}{\sqrt{\pi}} \int_0^x e^{-x^2} dx \quad \text{and } k \text{ is a constant.}$$

It seems to us that mathematically this method of approaching the problem is much less general, and much more difficult of application and of interpretation than our treatment which follows.

In our first paper we pointed out that:

We are convinced that equation (ix) represents no more than a first approximation to a true law of population growth. There are several characteristics of this curve which are too rigid and inelastic to meet the requirements of such a law. In (ix) the point of inflection must of necessity lie exactly half-way between the two asymptotes. Furthermore the half of the curve lying to the right of the point of inflection is an exact reversal of the half lying to the left of that point. This implies that the forces which during the latter part of the population history of an area act to inhibit the rate of population growth are equal in magnitude, and exactly similarly distributed in time, to the forces which in the first half of the history operate to accelerate growth. We do not believe that such rigid and inelastic postulates as these are, in fact, realized in population growth.

The same objections apply to the use of the equation of an autocatalytic reaction to the representation of organic growth in the individual. This fact has been noted by Robertson[14] who was the first to discover that, in general, growth follows much the same curve as that of autocatalysis. What needs to be done is to generalize (ix) in some such form as will free it from the two restrictive features (location of point of inflection and symmetry) we have mentioned, and will at the same time retain its other essential features.

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[13] Lehfeldt, R. A., The normal law of progress, *Jour. Roy. Stat. Soc.*, vol. 79, pp. 329–332, 1916

[14] *Loc. cit.*

We believe that we have now reached a solution, together with the additional feature of satisfying postulate 4 above. Before proceeding to the mathematical discussion it will be well to recall to mind, by figure 99, the general form and properties of equation (ix).

Considered generally, the curve

$$y = \frac{b}{e^{-ax} + c}$$

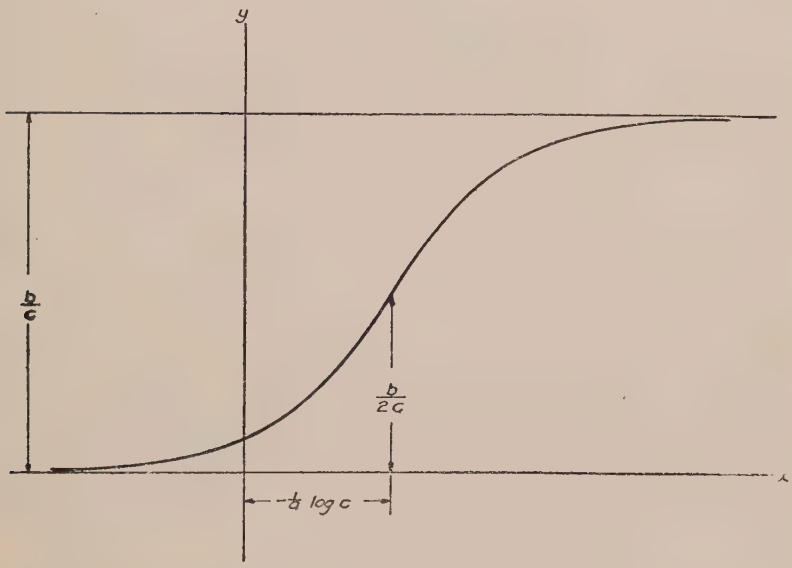

FIG. 99. GENERAL FORM OF CURVE GIVEN BY EQUATION (ix)

may be written

$$y = \frac{k}{1 + me^{ka'x}} \tag{x}$$

where

$$k = b/c, \quad m = 1/c, \text{ and } ka' = -a$$

Now the rate of change of y with respect to x is given by

$$\frac{dy}{dx} = -a'y\,(k - y)$$

or

$$\frac{\frac{dy}{dx}}{y\,(k - y)} = -a'. \tag{xi}$$

If y be the variable changing with time x (in our case population) (xi) amounts to the assumption that the time rate of change of y varies directly as y and as $(k-y)$, the constant k being the upper limit of growth, or in other words the value of the growing variable y at infinite time ($x = \infty$). Now since the rate of growth of y is dependent upon factors that vary with time we may generalize (xi) by using $f(x)$ in place of $-a'$, $f(x)$ being some as yet undefined function of time.

Then

$$\frac{dy}{y\,(k-y)} = f(x)\,dx,$$

whence

$$\frac{k-y}{my} = e^{-k\int f(x)\,dx}$$

and

$$y = \frac{k}{1+me^{-k\int f(x)dx}} = \frac{k}{1+me^{F(x)}} \qquad \text{(xii)}$$

where

$$F(x) = -k\int f(x)dx$$

Thus the assumption that the rate of growth of the dependent variable varies as (*a*) that variable, (*b*) a constant minus that variable, and (c) an arbitrary function of time, leads to equation (xii), which is of the same form as (ix), except that ax has been replaced by $F(x)$. If now we assume that $f(x)$ may be represented by a Taylor series, we have

$$y = \frac{k}{1+me^{a_1x+a_2x^2+a_3x^3+\ldots\ldots\ldots+a_nx^n}} \qquad \text{(xiii)}$$

If

$$a_2 = a_3 = a_4 = \ldots. = a_n = 0$$

then (xiii) becomes the same as (ix).

If m becomes negative the curve becomes discontinuous within finite time. Since this cannot occur in the case of the growth of the organism or of populations, nor indeed so far as we are able now to see, for any *phenomenal* changes with time, we shall restrict our further consideration of the equation to positive values only of m. Also since negative values of k would give negative values of y, which in the case of population or individual growth are, as has been pointed out already, unthinkable, we shall limit k to positive values.

With these limitations as to the values of m and k we have the following general facts as to the form of (xiii). y can never be negative, i.e., less than zero, nor greater than k. Thus the complete curve always falls between the x axis and a line parallel to it at a distance k above it. Further we have the following relations:

If

$$F(x) \doteq \infty \qquad y \doteq 0$$

$$F(x) \doteq -\infty \qquad y \doteq k$$

$$F(x) \doteq -0 \qquad y \doteq \frac{k}{1+m} \text{ from above}$$

$$F(x) \doteq +0 \qquad y \doteq \frac{k}{1+m} \text{ from below}$$

The maximum and minimum points of (xiii) occur where $\frac{dy}{dx} = 0$.

But

$$\frac{dy}{dx} = y(k-y)\cdot F'(x),$$

therefore we have maximum and minimum points wherever $F'(x) = 0$.

The fact that $\frac{dy}{dx} = 0$ when either $y = 0$ or $y - k = 0$ shows that the curve passes off to infinity asymptotic to the lines $y = 0$ and $y = k$.

The points of inflection of (xiii) are determined by the intersections of (xiii) with the curve

$$y = \frac{k}{2} - \frac{k}{2}\frac{F''(x)}{[F'(x)]^2} \tag{xiv}$$

Dropping all powers of x above the n^{th} we have two cases to consider, one where n is even and the other where n is odd. When n is even and a_n is positive the curve will be of the type shown in figure 100.

If a_n is negative the curve will be of the same form, except that it will be asymptotic to the line AB at both $x = +\infty$ and $x = -\infty$ and will lie between the line AB and the x axis.

If n is odd and a_n negative the curve will have the form shown in figure 101.

Since we are seldom justified in using over five arbitrary constants in any practical problem we may limit equation (xiii) still further by stopping at the third power of x. This gives the equation

$$y = \frac{k}{1 + me^{a_1x + a_2x^2 + a_3x^3}} \tag{xv}$$

If a_n is positive the curve of equation (xiii) is reversed and becomes asymptotic to the line AB, at $x = -\infty$ and to the x axis at $x = +\infty$. Thus in equation (xv) a_3 negative is a case of growth, and a_3 positive is a case of decay.

Equation (xv) has several special forms that are of interest, among them being a form similar in shape to the autocatalytic curve (i.e., with no maximum or minimum points and only one point of inflection) except that it is free from the two restrictive features mentioned above, that is, location of the point of inflection in the middle and symmetry of the two limbs of the curve. Asymmetrical or skew curves of this sort can only arise when

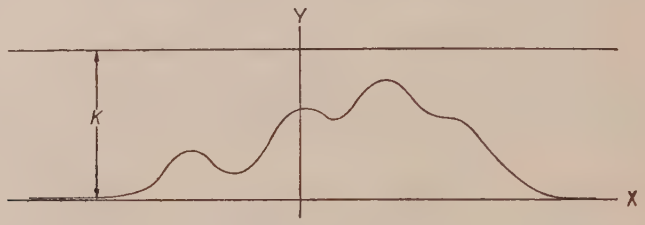

FIG. 100. SHOWING GENERAL FORM OF EQUATION (xiii) WHEN n IS EVEN AND a_n IS POSITIVE

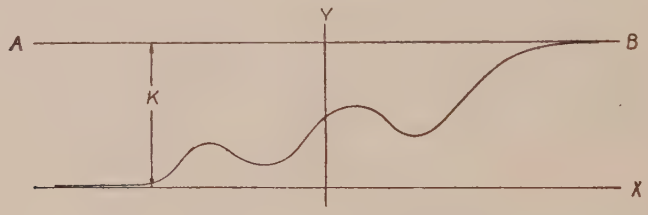

FIG. 101. GENERAL FORM OF EQUATION (xiii) WHEN n IS ODD, AND a_n IS NEGATIVE

equation (xiii) has no real roots. While any odd value of n may yield this form of curve the simplest equation that will do it is that in which $n = 3$, so that the equation of this curve becomes that of (xv).

An example of a curve of this type is shown in figure 102, the equation of this being

$$y = \frac{2}{1 + \cdot 1369\, e^{-x - .5x^2}} \qquad \text{(xvi)}$$

Using equation (xiv) we find that the points of inflection of (xvi) are the points of intersection of (xvi) and

$$y = 1 + \frac{12x}{(2 + 3x^2)^2} \qquad \text{(xvii)}$$

Having determined that the growth within any one epoch, or cycle may be approximately represented by equation (ix), or more accurately by (xv) the next question is that of treating several epochs or cycles. Theoretically some form of (xiii) may be found by sufficient labor in the adjustment of constants so that one equation with say 5 or 7 constants would describe a long history of growth involving several cycles. Practically, however, we have found it easier and just as satisfactory in other respects to treat each

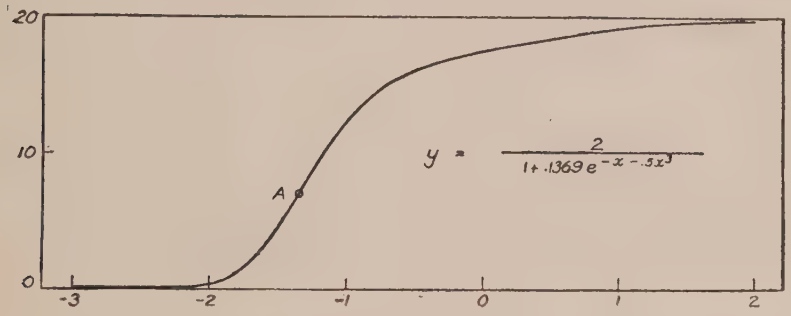

FIG. 102. SHOWING ONE FORM OF SKEW GROWTH CURVE

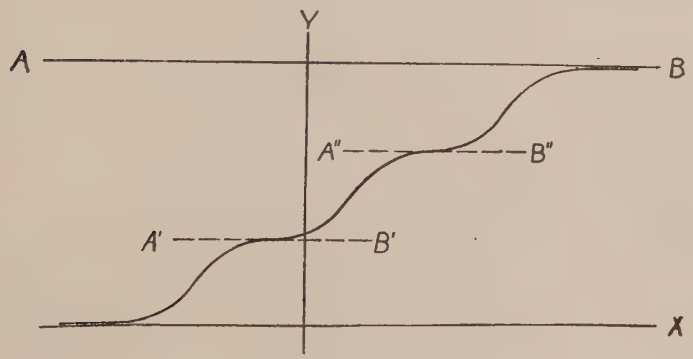

FIG. 103. GROWTH CURVE OF CYCLES, SUCCESSIVE IN TIME AND ADDITIVE

cycle by itself. Since the cycles of any case of growth are additive we may use for any single cycle the equation

$$y = d + \frac{k}{1 + me^{ka'x}} \qquad \text{(xviii)}$$

or more generally

$$y = d + \frac{k}{1 + me^{a_1x + a_2x^2 + a_3x^3}} \qquad \text{(xix)}$$

where in both of these forms d represents the total growth attained in all the previous cycles. The term d is therefore the lower asymptote of the cycle of growth under consideration and $d + k$ is its upper asymptote. The general picture of such a growth curve of several cycles will be like that shown in figure 103.

In treating any two adjacent cycles it should be noted that the lower asymptote of the second cycle is frequently below the upper asymptote of the first cycle due to the fact that the second cycle is often started before the first one has had time to reach its natural level. This for instance would be the case where a population entered upon an industrial era before the country had reached the limit of population possible under purely agricultural conditions.

Whenever the growth within the different cycles is symmetrical or nearly so there is considerable advantage in using equation (xviii) rather than (xix). Not only is the labor of fitting the curve less but the values of the constant a' will give the rates of growth of the different epochs.

FITTING THE CURVE

We have

$$y = \frac{k}{1 + me^{F(x)}}$$

$$me^{F(x)} = \frac{k - y}{y}$$

$$F(x) = \log (k - y) - \log m - \log y \qquad \text{(xx)}$$

In equation (xv)

$$F(x) = a_1x + a_2x^2 + a_3x^3$$

Let

$$\log m = a_0$$

Then

$$a_0 + a_1x + a_2x^2 + a_3x^3 = \log \frac{k - y}{y} \qquad \text{(xxi)}$$

Now if we choose 5 observed points and pass the curve through them we can determine the constants a_0, a_1, a_2, a_3, and k.

Assume as the coordinates of the 5 known points $(0, y_0)$, (x_1, y_1), (x_2, y_2), (x_3, y_3), (x_4, y_4)

Then

$$a_0 - \log \frac{k - y_0}{y_0} = 0 \qquad \text{(xxii)}$$

$$a_0 + x_1 a_1 + x_1^2 a_2 + x_1^3 a_3 - \log \frac{k - y_1}{y_1} = 0 \qquad \text{(xxiii)}$$

$$a_0 + x_2 a_1 + x_2^2 a_2 + x_2^3 a_3 - \log \frac{k - y_2}{y_2} = 0 \qquad \text{(xxiv)}$$

$$a_0 + x_3 a_1 + x_3^2 a_2 + x_3^3 a_3 - \log \frac{k - y_3}{y_3} = 0 \qquad \text{(xxv)}$$

$$a_0 + x_4 a_1 + x_4^2 a_2 + x_4^3 a_3 - \log \frac{k - y_4}{y_4} = 0 \qquad \text{(xxvi)}$$

After some rather lengthy but straightforward algebra, we get the following equation for the determination of k, on the assumption that the ordinates are equally spaced on the x axis,

$$y_1^4 y_3^4 (k - y_0) (k - y_2)^6 (k - y_4) = y_0 y_2^6 y_4 (k - y_1)^4 (k - y_3)^4 \qquad \text{(xxvii)}$$

To get the a's we proceed as follows.
Let

$$\beta_1 = \log \frac{k - y_1}{y_1} - \log \frac{k - y_0}{y_0}$$

$$\beta_2 = \log \frac{k - y_2}{y_2} - \log \frac{k - y_0}{y_0}$$

$$\beta_3 = \log \frac{k - y_3}{y_3} - \log \frac{k - y_0}{y_0}$$

Then we can express equations (xxii) to (xxvi) inclusive in terms of the a's and the β's.
We get

$$a_0 = \log \frac{k - y_0}{y_0} \qquad \text{(xxviii)}$$

$$a_1 = \frac{18\beta_1 - 9\beta_2 + 2\beta_3}{6x_1} \qquad \text{(xxix)}$$

$$a_2 = \frac{4\beta_2 - 5\beta_1 - \beta_3}{2x_1^2} \qquad \text{(xxx)}$$

$$a_3 = \frac{\beta_3 + 3\beta_1 - 3\beta_2}{6x_1^3} \qquad \text{(xxxi)}$$

This completes the solution of the problem of fitting, by the method here adopted.

The solution for the constants of the symmetrical form of growth curve may follow the lines of the preceding discussion. The symmetrical form, when written in the notation used above, becomes

$$y = \frac{b}{e^{-ax} + c} = \frac{k}{1 + me^{+ka'x}} = \frac{k}{1 + e^{a_0 + a_1 x}} \qquad \text{(xxxii)}$$

where

$$k = \frac{b}{c},\ m = \frac{1}{c} = e^{a_0},\ \text{and} - a = ka' = a_1$$

Assuming three points uniformly spaced along the x axis, as (o, y_o), (x_1, y_1) and $(2x_1, y_2)$, we have for the values of the constants

$$k = \frac{2y_0y_1y_2 - y_1^2(y_0 + y_2)}{y_0y_2 - y_1^2} \qquad \text{(xxxiii)}$$

$$a_0 = \log \frac{k - y_0}{y_0} \qquad \text{(xxxiv)}$$

$$a_1 = \frac{1}{x_1} \log \frac{y_0(k - y_1)}{y_1(k - y_0)} \qquad \text{(xxxv)}$$

These three equations furnish the constants for equation (xxxii) which may be written in any one of the given forms.

If a better fit is desired than that given by these selected points, we may use the following method of approximation.

Let α be an approximate value of a (or of $-a_1$ since $a = -a_1$).

Let h be the correction to α so that $a = \alpha + h$

Then equation (xxxii) may be written

$$y = \frac{b}{e^{-(\alpha + h)x} + c}$$

where b, c and h are the constants to be determined.

Rewriting the equation and expanding we have

$$y = \frac{b}{e^{-\alpha x} \cdot e^{-hx} + c} = \frac{b}{e^{-\alpha x}\left(1 - hx + \frac{h^2x^2}{\lfloor 2} - \frac{h^3x^3}{\lfloor 3} + \ldots\right) + c},$$

or approximately, since h is small,

$$y = \frac{b}{e^{-\alpha x}(1 - hx) + c},$$

from which

$$ye^{-\alpha x} - hxye^{-\alpha x} + cy - b = 0.$$

If now we proceed by the method of least squares and let r, the residual to be minimized, be the amount by which an observed point (x, y), fails to satisfy this equation, we have for the sum of the squares of the residuals

$$\Sigma r^2 = \Sigma\ (ye^{-\alpha x} - hxye^{-\alpha x} + cy - b)^2$$

Taking partial derivatives with respect to c, b and h, and equating to zero, we have the following equations for the determination of the constants.

$$b\,n - c\Sigma y + h\Sigma xye^{-\alpha x} = \Sigma ye^{-\alpha x} \qquad \text{(xxxvi)}$$

$$b\Sigma y - c\Sigma y^2 + h\Sigma xy^2e^{-\alpha x} = \Sigma y^2e^{-\alpha x} \qquad \text{(xxxvii)}$$

$$b\Sigma xye^{-\alpha x} - c\Sigma xy^2e^{-\alpha x} + h\Sigma x^2y^2e^{-2\alpha x} = \Sigma xy^2e^{-2\alpha x} \qquad \text{(xxxviii)}$$

To illustrate the use of the preceding theory we will proceed to adjust, by this method of approximation, the equation for the population of the United States as given in our first paper,[15] this equation having been fitted by three selected points. The equation as originally given was

$$y = \frac{2.930301}{e^{-0.0313395x} + 0.014854}$$

in which y represents the population in millions of persons and x represents the number of years beyond 1780. Using this value of a (0.0313395) as our approximate value, α, we proceed to form (see table 172) the sums needed in equations (xxxvi) (xxxvii) and (xxxviii).

From the totals in this table we may write the equations

$$\begin{aligned} 13b - 430.314\,c + 2017.8489\,h &= 31.541177 \\ 430.314b - 24371.220\,c + 84751.323\,h &= 895.32051 \\ 2017.8489b - 84751.323\,c + 365945.80\,h &= 4633.2505 \end{aligned}$$

Solving these equations we find

$$\begin{aligned} b &= 2.90688 \\ c &= 0.014786 \\ h &= 0.0000567 \end{aligned}$$

Thus the new value of a is

$$a = 0.0313395 + 0.0000567 = 0.0313962$$

[15] Pearl, R., and Reed, L. J., *loc. cit.*

TABLE 172

Computation of curve for United States

x	y	$e^{-\alpha x}$	y^2	$xye^{-\alpha x}$	$xy^2e^{-\alpha x}$	$xy^2e^{-2\alpha x}$	$x^2y^2e^{-2\alpha x}$	$ye^{-\alpha x}$	$y^2e^{-\alpha x}$
10	3.929	0.730961	15.437	28.7195	112.860	82.4965	824.97	2.871946	11.28384
20	5.308	0.534304	28.175	56.7217	301.080	160.8684	3217.37	2.836086	15.05396
30	7.240	0.390555	52.418	84.8286	614.148	239.8585	7195.75	2.827618	20.47196
40	9.638	0.285481	92.891	110.0586	1060.733	302.8192	12112.77	2.751751	26.51862
50	12.866	0.208675	165.534	134.2406	1727.140	360.4114	18020.57	2.684813	34.54281
60	17.069	0.152533	291.351	156.2152	2666.429	406.7182	24403.09	2.603586	44.44061
70	23.192	0.111496	537.869	181.0071	4197.914	468.0522	32763.65	2.585815	59.97023
80	31.443	0.081499	988.662	205.0073	6446.048	525.3515	42028.12	2.562592	80.57557
90	38.558	0.059572	1486.719	206.7304	7971.107	474.8595	42737.35	2.297004	88.56789
100	50.156	0.043545	2515.624	218.4058	10954.360	477.0101	47701.01	2.184058	109.54362
110	62.948	0.031829	3962.451	220.3991	13873.686	441.5982	48575.80	2.003629	126.12441
120	75.995	0.023266	5775.240	212.1756	16124.285	375.1538	45018.46	1.768130	134.36904
130	91.972	0.017006	8458.849	203.3394	18701.533	318.0530	41346.89	1.564149	143.85795
Total.....	430.314		24,371.220	2,017.8489	84,751.323	4,633.2505	365,945.80	31.541177	895.32051

and the new equation is

$$y = \frac{2.90688}{e^{-0.0313962x} + 0.014786} \qquad \text{(xxxix)}$$

Computing the values of y for the census years and forming the deviations of observed from computed, we have as the root mean squared residual 0.448 as against 0.463 for the original equation, indicating a slightly better fit.

It should be noted that in using this approximation the value α may be obtained by any method desired, the only requirement being that it shall be large as compared with h. Usually the best results are obtained when we use three selected points to determine α, these points being chosen to indicate the trend but not necessarily being any one of the observed points.

PROBABLE ERRORS

H. S. Reed,[16] in his study of the simple autocatalytic growth curve has shown that in the growth of the sunflower the variation of the observations about their means lying along the curve is proportional, in a remarkably constant way, to the absolute values of the successive means. This is what might reasonably be expected.

In addition to this element in the case, it is apparent that the accuracy of determination of the upper asymptote k of the curve will depend in part directly upon the *number* of observed ordinates from which the fitting must be done, and their location relative to the whole growth cycle. Thus if all the observed points lie in the first half of the curve (below the point of inflection) we shall evidently get a less reliable estimate of the upper asymptote than if the observations cover say three fourths or more of the whole cycle. This point will be apparent from examples presented in the next chapter of this book.

There are several ways in which the problem of the probable errors of the constants of the single cycle form of (xiii)

$$y = \frac{k}{1 + me^{-a_1 x}} \qquad \text{(xl)}$$

may be approached.

Assuming that the graduation of a series of observed points by (xl) may be regarded as a "false position" in the sense of Pearson's classical paper

[16] Reed, H. S., Growth and variability in *Helianthus*, *Amer. Jour. Bot.*, vol. 6, pp. 252–271, 1919.

in the *Philosophical Magazine* on this application of the method of least squares, we may take residuals of the form:

$$\Delta y = \frac{y}{k}\Delta k + \frac{m}{k}y^2 x e^{-a_1 x}\Delta a_1 - \frac{1}{k}y^2 e^{-a_1 x}\Delta m \qquad \text{(xli)}$$

which leads, by ordinary least square procedure, to normal equations as follows:

$$\begin{aligned}
&\frac{1}{k}\Sigma y^2\Delta k + \frac{m}{k}\Sigma y^3 x e^{-a_1 x}\Delta a_1 - \frac{1}{k}\Sigma y^3 e^{-a_1 x}\Delta m = \Sigma y\Delta y,\\
&\frac{1}{k}\Sigma y^3 x e^{-a_1 x}\Delta k + \frac{m}{k}\Sigma y^4 x^2 e^{-2a_1 x}\Delta a_1 - \frac{1}{k}\Sigma y^3 x e^{-2a_1 x}\Delta m = \Sigma y^2 x e^{-a_1 x}\Delta y, \qquad \text{(xlii)}\\
&\frac{1}{k}\Sigma y^3 e^{-a_1 x}\Delta k + \frac{m}{k}\Sigma y^4 x e^{-2a_1 x}\Delta a_1 - \frac{1}{k}\Sigma y^4 e^{-2a_1 x}\Delta m = \Sigma y^2 e^{-a_1 x}\Delta y.
\end{aligned}$$

Or, in view of H. S. Reed's (*loc. cit.*) results, we may, with perhaps better logic and common sense take *relative* residuals of the form $\Delta y/y$, rather than absolute residuals. We then have

$$\frac{\Delta y}{y} = \frac{1}{k}\Delta k + \frac{m}{k}xye^{-a_1 x}\Delta a_1 - \frac{1}{k}ye^{-a_1 x}\Delta m, \qquad \text{(xliii)}$$

which leads to normal equations as follows:

$$\begin{aligned}
&\frac{n}{k}\Delta k + \frac{m}{k}\Sigma xye^{-a_1 x}\Delta a_1 - \frac{1}{k}\Sigma ye^{-a_1 x}\Delta m = \Sigma\frac{\Delta y}{y},\\
&\frac{1}{k}\Sigma xye^{-a_1 x}\Delta k + \frac{m}{k}\Sigma x^2 y^2 e^{-2a_1 x}\Delta a_1 - \frac{1}{k}\Sigma xy^2 e^{-2a_1 x}\Delta m = \Sigma xye^{-a_1 x}\frac{\Delta y}{y}, \qquad \text{(xliv)}\\
&\frac{1}{k}\Sigma ye^{-a_1 x}\Delta k + \frac{m}{k}\Sigma xy^2 e^{-2a_1 x}\Delta a_1 - \frac{1}{k}\Sigma y^2 e^{-2a_1 x}\Delta m = \Sigma ye^{-a_1 x}\frac{\Delta y}{y}.
\end{aligned}$$

So far we have gone on an assumption of the original three constant form of the curve (xl). Actually in most of our fitting work now, we use the four constant curve, of the form

$$y = d + \frac{k}{1 + me^{-a_1 x}}. \qquad \text{(xlv)}$$

Because of the fact that in practice we have found d to be generally very small in magnitude, and for the sake of simplicity in the analysis, we may write (xliii) and (xliv) in the same form as above, except that $\Delta y/y$ is replaced by $\Delta y/(y - d)$. This is in effect disregarding variations

in d. We feel this, for reasons stated above, to be justifiable in a first approach to the probable errors of these curves.

By the application of (xliv) it is possible to get, by the well-known procedure employed by Pearson in his many studies of probable errors, the standard errors (and thence the probable errors) of k, a, and m in terms of the standard error of $\Delta y/y$ or $\Delta y/(y - d)$.

The probable errors so obtained will measure the fluctuations due to the sampling of theoretical curves of the form (xlv) or (xl) against a definite set of observed population points given by census counts. This would appear to be the *kind* of probable error which is desirable in the premises. The problem of fluctuations in census counts themselves is quite a different one, and one about which we are not directly concerned in a strictly *curve fitting* enterprise, which is what we are discussing in our application of the population curve (xiii).

In the next chapter these probable error equations will be applied to specific numerical examples. It will be found that where there are a reasonable number of fairly accurate census counts to serve as the basis of the curve fitting the probable errors are small, both absolutely and relatively. Of course in cases where the observed points are few in number, or are at the extreme ends, or at only a short portion of the center of the cycle, or show large deviations from the theoretical curve, the probable errors will be larger. We have pointed out specifically in earlier papers on the subject that this would be the fact in some particular curves which we have presented.

Finally, it should be noted that the values given by the procedure here followed are to be regarded as close approximations only to the true values of the probable errors, because in our work the correlations between the errors of the several constants have been neglected. We are confident, however, that the inclusion of the additional terms involving these correlations would not sufficiently alter the final result to change any conclusion that would ever be drawn from the curves in practical statistical work.

CHAPTER XXV

The Growth of Human Population[1]

It is a sound generalization of experience that the best proof of any pudding lies in the eating of it. Owing to pressure of other work we have hitherto published only scant evidence of the effectiveness of the curve of population growth set forth in Chapter XXIV. We have ourselves known from actual trial for a number of years that this curve described with extraordinary exactness the growth of population in a large proportion of all the countries for which any population data exist, and have on that account been able to bear with some equanimity the slightly premature and sweeping criticism that the curve had no relation to population growth. It is hoped that the evidence now to be presented will indicate the validity and generality of our curves for human population growth.

The plan of this chapter is as follows: To present first the population data and the fitted curve for the United States, and some deductions therefrom, as a sort of paradigm. This will be followed by a presentation in the briefest form of observed populations from census counts for 12 countries of Europe, and the curves fitted to them. The following section will deal with three countries in the Far East in a similar way. The next section will discuss the population of the world, and finally a section will be devoted to the discussion of the growth of city populations.

From this outline it will be perceived that we have applied our hypothesis to the most diverse population groups. The diversity is of many sorts including extreme racial differences; differences of cultural development, all the way from low agricultural and even hunting stages to the most highly developed urban industrialization; differences in respect of migration in and out of the group dealt with; and differences of absolute size. Furthermore we have in two cases dealt with countries which within the period of recorded population history are known to have passed from one stage of culture into another. If then, as will presently be demonstrated, our hypothesis is able to describe with great accuracy the growth of populations so diverse as these,

[1] This chapter is by Raymond Pearl and Lowell J. Reed. The material in it has not hitherto been published, except that the application of the curve of population growth was first made in our original paper (in preceding chapter) to the United States, and in *The Biology of Death*, Pearl presented graphs for the population growth of Servia and France.

we think it may fairly be claimed that we have established at least a strong presumption that our basic hypotheses are at least in accord with what goes on in nature. We shall present here a series of facts, capable of verification by anyone who can read and carry out computations of no great degree of complexity. We are entirely content to let these facts speak for themselves.

We are of the opinion, again based upon rather extensive experience, that this curve will be found useful in dealing with many other sorts of phenomena changing in time, in addition to population growth. But we have no desire or intention at this time to press this aspect of the matter. On the contrary we propose to confine the present discussion exclusively to population growth, leaving it to others, and possibly to ourselves at some future time, to discuss applications of this curve outside the population field.

It should be understood that the limitations of our claims regarding this curve are clear and distinct. We think that the evidence to be presented strongly suggests that it may be regarded as an adequate description of the phenomena of population growth. It seems to us fairly to correspond, in a modest way, to Kepler's Law of the motion of the planets in elliptic orbits, but to lack the heuristic element which Newton added in showing that gravitation would account for elliptic orbits; or to Boyle's Law prior to Clerk Maxwell's kinetic theory. In short, nothing in the mathematics of Chapter XXIV or their successful application in this chapter gives the slightest inkling of the nature of the causes lying behind postulate 5 (page 568) of our descriptive curve. The one real step in the direction of the discovery of these causes which has so far been made, is the demonstration[2] that these causes are not things peculiar to human beings, such as the economic or social structure or organization of human society. This is proved by the experimental demonstration that the same curve describes with precision the growth of population of the fruit fly *Drosophila melanogaster*. This means that the search must be thrown back to more fundamental natural causes, biological, physical or chemical. It seems probable that the hopeful direction of further research in this field is not along the statistical pathway with human population as material, but along the experimental, where populations of lower organisms can be studied under controlled conditions.

THE METHOD OF PRESENTING THE EVIDENCE

A preliminary word of explanation is desirable as to the manner in which the evidence about population growth will uniformly be presented in what follows. For each population group one or another form of the general curve

[2] See Pearl, R., *The Biology of Death*, pp. 253–254.

presented in Chapter XXIV has first been fitted by methods there described to the known (or estimated) data as to the actually existing populations at different dates. The actual equation found by this fitting process is presented. In a table are given (*a*) each observed population, to the nearest thousand, against the date of its observation; and (*b*) the population calculated from the curve for the same date. All populations are stated as in millions, with three decimal places, thus making the actual statement, as already noted, to the nearest thousand. This is certainly all the accuracy that the census method warrants and for some countries and some times probably more.

Now if, as we believe to be the case, this curve states a first approximation to a descriptive law of nature, there is some justification for extrapolation in both directions from the period of known population history. That is to say, the curve enables and in some degree justifies the prediction of future populations, and the estimation of past populations before census counts were made. Accordingly we have in each case extended the curves to the upper and the lower asymptotic conditions realizing that in so doing we are inviting criticism.

The data of each table are presented graphically in a diagram. These diagrams are all prepared on the following uniform plan. An observed population is plotted as a small circle. The curve in that portion of its course comprised within the dates of observed populations is plotted as a continuous heavy curved line. Outside these dates the extrapolated portions of the curve are plotted as broken dash lines.

In order to forestall perhaps in some degree the criticism which will be directed towards the extrapolated portions of the curve some further explanation may be given. There are three points which should be comprehended with the utmost clearness, and kept constantly in mind in viewing the collection of evidence to be presented. These are:

1. The extrapolated[3] portions of the curve have no bearing whatsoever upon the question of the adequacy of the curves to describe the *known facts*

[3] For the aid and comfort of the non-mathematical reader two technical terms which will be frequently used in what follows may be briefly explained. "Extrapolation" (pronounced, at least by most American mathematicians, with a distinct accent on the syllable "trap" which is a quite unintentional and unconscious bit of humor, because the "trap" in any extrapolation may be a large and vicious one) means the extension of a theoretical curve into regions beyond that in which lie the observations upon which the theoertical curve is based. "Asymptote" means a limiting value towards which a curve approaches ever more and more closely but never quite reaches. The series $\frac{1}{2} + \frac{1}{4} + \frac{1}{8} + \frac{1}{16} + \frac{1}{32} + \frac{1}{64} +$ etc., has as its asymptotic limit unity (1.00 . . . .), but no matter how far the series is continued it will always sum up to something less than 1.

of population growth. It is upon the success or failure of the attempt to demonstrate this adequacy, *and upon this alone,* that the judgment of the scientific validity of the hypothesis must rest.

2. The prediction of future populations by extrapolation is wholly dependent upon and always carries with itself the assumption that in the period forward from the latest observed population datum to the approximate attainment of the upper asymptotic population *no fundamentally new factor or forces influencing the rate of population growth different from those which have operated during the known historical period of this population's growth shall come into play.* In short, the prediction is always based upon the assumption that the conditions which led to the law according to which that particular growth has occurred in the known past will continue to operate.

What do we mean by such new factors or forces? The only ones we have found any evidence for the actual occurrence of in our extensive study of population are those implied in the passage of a group of people from one stage or epoch of culture to another, as from the agricultural to the industrial; or, in the case of small countries essentially catastrophic occurrences such as change of boundaries by war, extensive famine, or emigration in a short time period of a large fraction of the total population. Normal, non-catastrophic migratory movements, as we shall see, have no appreciable effect on population growth; a phenomenon, by the way, with which Benjamin Franklin was entirely familiar, and indeed stated a long time ago.

Such a restrictive assumption as 2 is of course implicit in all descriptive laws of nature. The statement that the earth will describe a particular, now predictable, elliptic orbit about the sun in the year 2196 A.D. is conditional on several assumptions: one that in that year the whole solar system in its flight through space will not crash into some other sidereal wanderer; another that in the period from 1924 to 2196 no general or particular rearrangement of the heavenly bodies in our more or less immediate neighborhood shall have occurred, consequent upon the particular gravitational field dominated by the sun having picked up and gathered into itself any mass of matter, cometary or other, sufficiently large to cause new perturbations of the planetary orbits.

3. The determination of asymptotic populations by this curve is subject to a probable error. An approximate analytical expression for this probable error is given in Chapter XXIV. The accuracy of determination of an extrapolated ordinate will vary inversely as some function of the extent of the extrapolation. For example, to take an extreme case it is obvious that one could estimate much more accurately the probable population of a given area ten years in the future, from a curve based upon knowledge of the actual

growth of the same population for two hundred years in the past, than he could the probable population two hundred years in the future from a knowledge of its actual growth during only fifty to one hundred years in the past. Now actually something like this last condition is what confronts us in calculating the upper asymptote of most of the populations in the present cycle of their growth. Few countries have dependable census records for more than a century past; many do not even go back that far. But, as we shall presently see, most countries are certainly in the first half of the current cycle of growth. This means that in predicting the upper asymptote we are extrapolating the curve something like two hundred years into the future. This is much too far to be expected to give entirely accurate results We have much evidence that our curve will predict population values for ten to thirty years with great accuracy. And in some cases, as will be seen, we can from the nature of phenomena predict the upper asymptotic population with a high probability of accuracy. But in general we wish to emphasize that *predicted asymptotic populations are subject to a probable error.*

The proper thing to do, of course, in regard to predictions of population growth, as well as for astronomical predictions, is to revise the prediction whenever additional data are provided. Specifically the population curve should be refitted every time a new census is taken. All that the present extrapolations say is that, on the basis of existing knowledge at the time the fits were made, the probable future growth of any particular population will be so and so, subject to a probable error which may be large. The next census will in some degree, greater or less, alter this prediction, but it will do so because the body of existing knowledge has become greater. Such a constant revision of future estimates is particularly necessary in the case of population growth, because of the numerous factors, social, economic and others, which may cause perturbations in the normal course of events.

All of the above considerations apply with perhaps even greater force to extrapolation back from the date of the earliest census record to the lower asymptotic population. In all of the countries of Europe certainly, the present epoch or cycle of population growth starts from a lower asymptote which represents the upper limit of the preceding cycle or epoch. Some critics who have not looked into the implications and meaning of our hypothesis with sufficient care have criticized the general curve on the ground that extrapolated backwards the curve of a particular country for the present cycle did not give reasonable values for the population at some date in the preceding epoch or cycle. Of course it does not. The only reason we do not fit curves to describe these cycles preceding the one we are now in

is simply because reliable observed data are in nearly every case lacking. For only two countries, Japan and Germany, have we been able to get observed figures of population falling in two different growth cycles. These cases are dealt with below.

The immediate source of the observed figures is indicated in each case. Mostly they have been taken from the handiest derivative source, such as the Statesman's Year Book or Knibbs's very valuable treatise on population. Pains have been taken to check these figures, by a process of random sampling, against originals where possible, but every single observed figure has not been verified in this way. It would be impossible to do so with the bibliographic resources at our command. The sources we have used, however, are in general known by statisticians to be reliable.

In all the curves y denotes calculated population in millions, and x time in base units of ten years taken from 1800 as origin.

THE GROWTH OF THE POPULATION OF THE UNITED STATES

The observed and calculated populations for the United States are given in table 173. The calculations are based upon census data from 1790 to 1910 inclusive.

The equation to the calculated curve (United States) is

$$y = \frac{197.27}{1 + 67.32\, e^{-0.0313x}} \qquad \text{(i)}$$

It should be noted that the period of observations lies below the point of inflection of the calculated curve for the present cycle of growth of the population of the United States, and covers roughly the latter two-thirds of the first half of the whole theoretical curve.

The data of table 173 are shown graphically in figure 104.

It is obvious that there is extremely close agreement between observation and theory throughout the period of one hundred twenty years covered by census data. A closer fit could hardly be hoped for.

Let us examine some further consequences which flow from equation (i). The first question which interests one is this: when did or will the population curve of this country pass the point of inflection, and exhibit a progressively diminishing instead of increasing rate of growth? It is easily determined that this point occurred about April, 1914, on the assumption that the numerical values of (i) reliably represent the law of population growth in this country. In other words, so far as we may rely upon present numerical values, the United States has already passed its period of most rapid popula-

tion growth, unless there comes into play some factor not now known and which has never operated during the past history of the country to make the rate of growth more rapid. This latter contingency is improbable. The 1920 census confirms the result indicated by our curve, that the period of most rapid population growth was passed somewhere in the last decade. The population at the point of inflection works out to have been 98,637,000, which was in fact about the population of the country in 1914.

The upper asymptote given by (i) has the value 197,274,000 roughly. This means that according to equation (i) the maximum population which

TABLE 173

*United States**

YEAR	POPULATION IN MILLIONS		YEAR	POPULATION IN MILLIONS	
	Calculated	Observed		Calculated	Observed
Lower asympt.	0.000		1900	76.870	75.995
1700	0.239		1910	91.972	91.972
1720	0.446		1920	107.394	
1740	0.833		1930	122.397	
1760	1.553		1940	136.318	
1780	2.887		1950	148.678	
1790	3.929	3.929	1960	159.230	
1800	5.336	5.308	1970	167.945	
1810	7.228	7.240	1980	174.941	
1820	9.757	9.638	1990	180.437	
1830	13.109	12.866	2000	184.678	
1840	17.506	17.069	2020	190.341	
1850	23.192	23.192	2040	193.509	
1860	30.412	31.443	2060	195.249	
1870	39.372	38.558	2080	196.337	
1880	50.177	50.156	2100	196.681	
1890	62.769	62.948	Upper asympt.	197.274	

*Data from *Statistical Abstract.*

continental United States, as now areally limited, will have in its present growth cycle will be roughly twice the present population.

It will at once be pointed out that many European countries have a much greater density of population than 66 persons to the square mile, as for example, Belgium with 673, Netherlands with 499, etc. But it must not be forgotten that these countries are far from self-supporting in respect of physical means of subsistence. They are economically self-supporting, which is a very different thing, because by their industrial development at home and in their colonies they produce money enough to buy physical

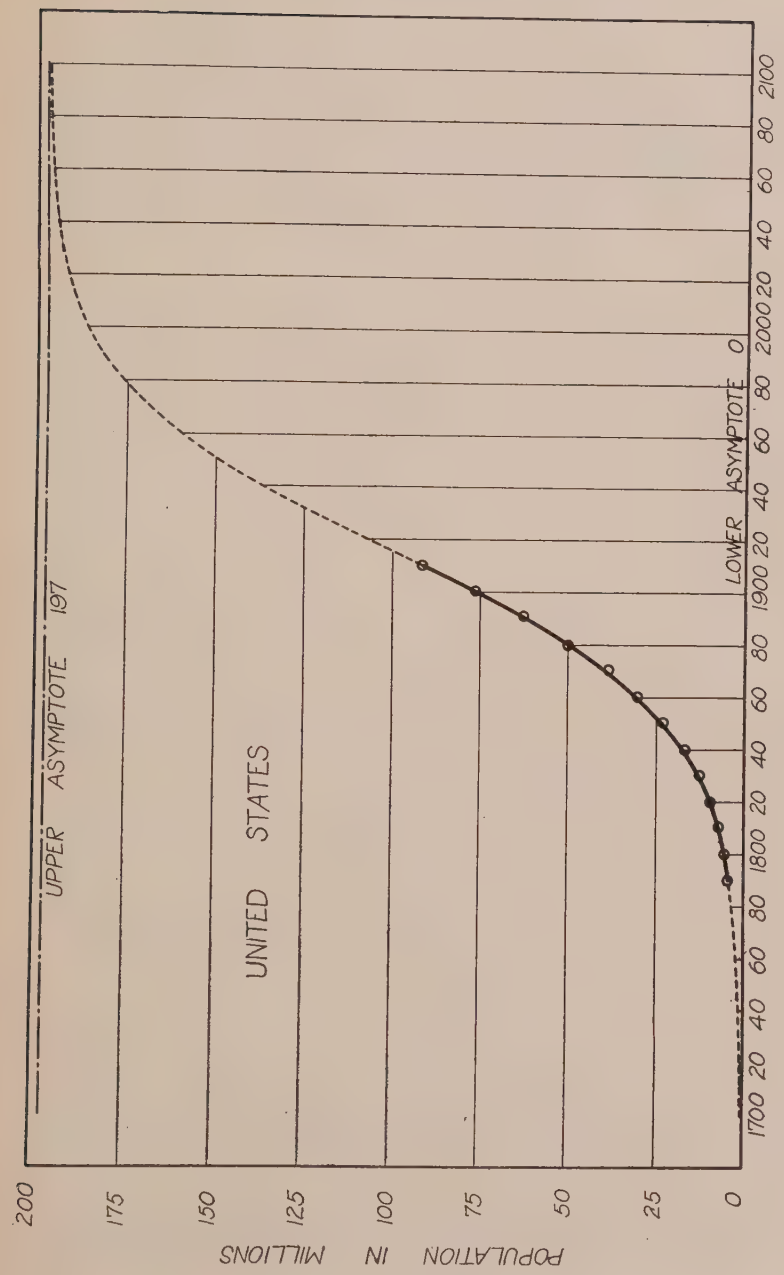

FIG. 104. THE POPULATION GROWTH OF THE UNITED STATES

means of subsistence from less densely populated portions of the world. We can, of course, do the same thing, provided that by the time our population gets so dense as to make it necessary there still remain portions of the globe where food, clothing material, and fuel are produced in excess of the needs of their home population. But in this, and in any other scientific discussion of population, it is necessary to limit sharply the area one is to talk about. We are here dealing with population, and by direct implication the production of physical means of subsistence, within the present area of continental United States.

Now, 197,000,000 people will require, on the basis of our present food habits[4] about 260,000,000,000,000 calories per annum. The United States, during the seven years 1911 to 1918, produced as an annual average, in the form of human food, both primary and secondary (i.e., broadly vegetable and animal), only 137,163,606,000,000 calories per year.[4] So that unless our food habits radically change, and a man is able to do with less than 3000 to 3500 calories per day, or unless our agricultural production radically increases,[5] it will be necessary when our modest figure for the asymptotic population is reached, to import nearly or quite one-half of the calories necessary for that population. It seems improbable that the population will go on increasing at any very rapid rate after such a condition is reached. And is it at all reasonable to suppose that at such time, with all the competition for the means of subsistence which the already densely populated countries of Europe will be putting up, there can be found any portion of the globe producing food in excess of its own needs to an extent to make it possible for us to find the calories we shall need to import?

Altogether, we believe it will be the part of wisdom for anyone disposed to criticize our asymptotic value of $197\frac{1}{4}$ millions because it is thought too small, to look further into all the relevant facts.

But, as we have emphasized above, the upper asymptotic figure has a probable error. The constants, with their probable errors are:

$$k = 197.27 \pm 0.55 \text{ millions of population}$$
$$a_1 = 0.03134 \pm 0.00013$$
$$m = 67.32 \pm 0.17$$

These are small probable errors, both absolutely and relatively, but there must always be remembered the possibility of systematic change implied in the second assumption stated in the introduction (page 587).

[4] *Cf.* Chapter XIV above.

[5] In this connection one should study East's masterly analysis of potential agricultural development in his brilliant book *Mankind at the Cross Roads*, New York (Scribner's), 1923.

THE GROWTH OF EUROPEAN POPULATIONS

Austria

The observed and calculated populations of Austria are given in table 174, and graphically in figure 105. The equation to the fitted curve is:

$$y = 12.523 + \frac{35.037}{1 + 22.765\, e^{-0.0268x}} \qquad \text{(ii)}$$

TABLE 174

*Austria**

YEAR	POPULATION IN MILLIONS		YEAR	POPULATION IN MILLIONS	
	Calculated	Observed		Calculated	Observed
Lower asympt.	12.523		1910	28.478	28.570
1700	12.628		1920	30.823	
1720	12.703		1930	33.137	
1740	12.829		1940	35.344	
1760	13.042		1950	37.381	
1780	13.401		1960	39.202	
1800	13.997		1970	40.785	
1810	14.426		1980	42.133	
1820	14.969		1990	43.252	
1830	15.653	15.590	2000	44.165	
1840	16.506	16.580	2020	45.490	
1850	17.553	17.530	2040	46.316	
1860	18.821		2060	46.823	
1869	20.163	20.390	2080	47.124	
1880	22.070	22.140	2100	47.303	
1890	24.037	23.900	Upper asympt.	47.560	
1900	26.193	26.150			

* Observed populations taken from Knibbs.

It will be noted that the observed points lie below the point of inflection of the theoretical curve for the present cycle of Austria's population growth, and roughly in the second quarter of the curve's whole course.

The graduation is obviously excellent within the period covered by the known data. The curve starts from a lower asymptote of roughly $12\frac{1}{2}$ millions. This suggests the starting of the present cycle of population growth in Austria sometime probably between 1650 and 1750 from the top or near the top of a prior cycle.

The extrapolation of the curve beyond 1910 has obviously no significance beyond showing what would have been the probable population growth of

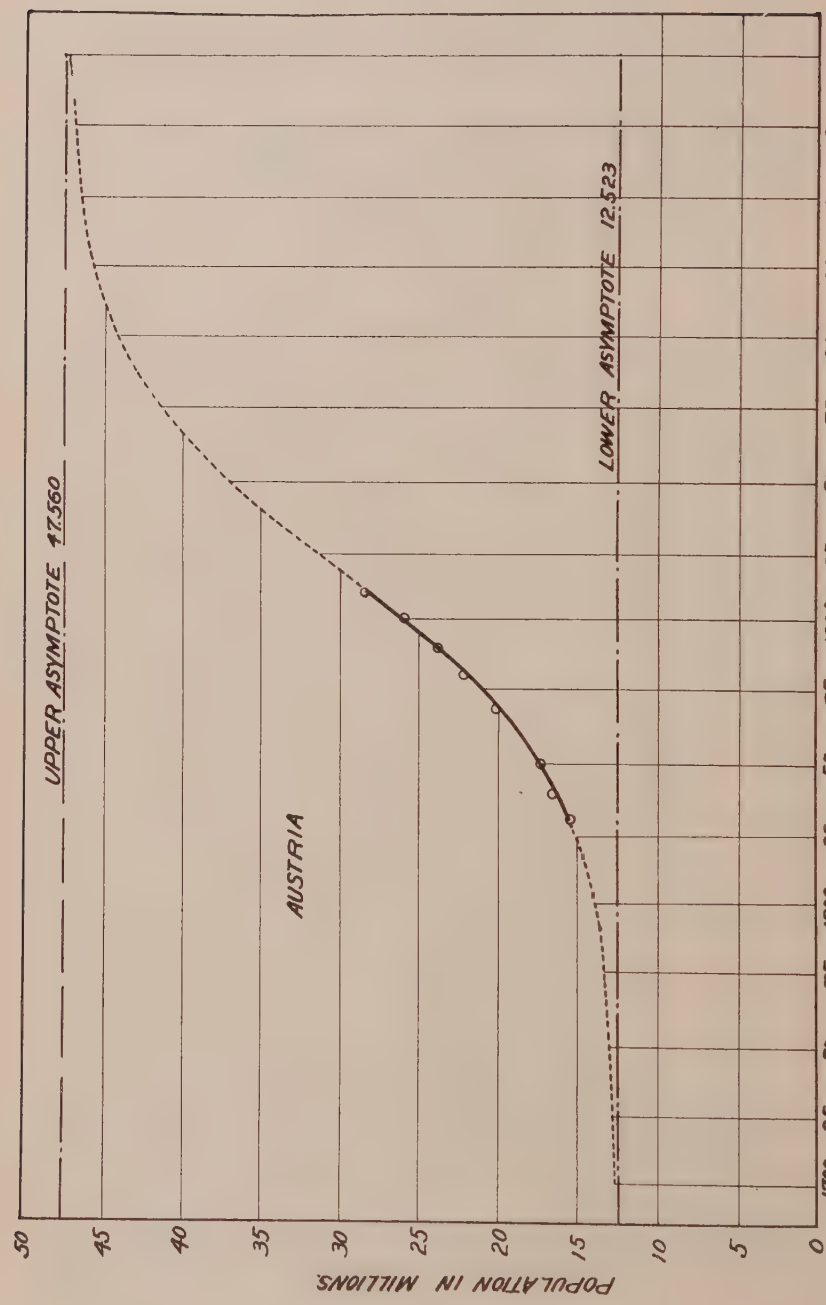

FIG. 105. THE POPULATION GROWTH OF AUSTRIA

Austria as it existed in 1910, if nothing relating to the country had been fundamentally altered. But in 1914 to 1918, and subsequently at Versailles, a good deal about Austria *was* fundamentally altered ,including her boundaries. Hence the old curve cannot apply in the future. We must wait for two censuses at least under the new conditions to get data upon which to calculate the perturbation of the population curve produced by the war, and the probably even more disturbing peace which followed it.

Examining the extrapolation of the curve backward we note that the basis of experience (known populations) covers eighty years. The curve indicates that in 1700 the lower asymptote was fairly closely approached, the difference being 105,000 people. We are thus extrapolating backwards one hundred thirty years from an eighty year experience base. This gives an extrapolation ratio (8:13) to the lower asymptote almost identical with that of the United States (12:19) to the upper asymptote.

Belgium

Table 175 gives the observed and calculated values for the population growth of Belgium. The equation to the fitted curve is:

$$y = 3.410 + \frac{9.994}{1 + 43.688\, e^{-0.0307x}} \qquad \text{(iii)}$$

The data of table 175 are shown graphically in figure 106.

Again the fit within the observation period is obviously almost perfect. The curve starts from an estimated lower asymptote of just under $3\frac{1}{2}$ millions, indicating a prior growth cycle which brought the population up to about that figure. Unfortunately we cannot use the earlier records of Belgian population because of different boundaries than those of the present era.

The indicated upper asymptotic population is nearly $13\frac{1}{2}$ millions. This is roughly double the present population. It implies a density of over 1300 to the square mile. Probably it will never be attained. It seems not unlikely that as world population pressure increases a country like Belgium, which can only support its present population by exchanging manufactured goods for means of subsistence produced in less densely populated parts of the world, will show a skew or asymmetrical curve of population growth, like figure 102, with a smaller value for the upper asymptote than that given by a symmetrical growth curve. In any case a few more censuses should give indication of this tendency if it is to appear.

It will be noted that Belgium's known population covers about the same portion of the whole present cycle as does that of Austria. Actually the

basis of known experience covers only sixty-four years, while if we regard the population at 2100 as sufficiently close to the asymptotic population, we are extrapolating one hundred ninety years. This gives an extrapolation ratio of 64 : 190 or approximately 1:3 to the upper asymptote. This certainly implies a considerable probable error in the result. Similar considerations apply covering the lower asymptotic figure of $3\frac{2}{5}$ millions. Here we are extrapolating one hundred forty-six years on a basis of sixty-four years experience.

TABLE 175

*Belgium**

YEAR	POPULATION IN MILLIONS		YEAR	POPULATION IN MILLIONS	
	Calculated	Observed		Calculated	Observed
Lower asympt.	3.410		1890	6.070	6.069
1700	3.421		1900	6.710	6.694
1720	3.430		1910	7.427	7.424
1740	3.446		1920	8.148	
1760	3.477		1940	9.679	
1780	3.532		1960	10.972	
1800	3.634		1980	11.922	
1820	3.816		2000	12.543	
1840	4.134		2020	12.919	
1846	4.272	4.337	2040	13.136	
1856	4.546	4.529	2060	13.257	
1866	4.894	4.828	2080	13.324	
1876	5.325	5.336	2100	13.360	
1880	5.515	5.520	Upper asympt.	13.404	

* Observed values are census counts, taken from the *Statesman's Year Book*. Early population figures for Belgium are on a different area and are not comparable with the later census values, nor with the extrapolated curve calculated from these later figures.

Denmark

The data for the population growth of Denmark, observed and calculated are set forth in table 176. The equation of the calculated curve is:

$$y = 0.672 + \frac{5.64}{1 + 20.870\, e^{-0.0230x}} \qquad \text{(iv)}$$

The fit of theory to observations is well-nigh perfect. The curve starts from a lower asymptote of 672,000 population, and had attained 699,000 by the year 1700. The prior cycle was presumably a long flat curve. The upper asymptote of the curve is approximately $6\frac{1}{4}$ millions, a not unreasonable figure, but still subject to a considerable probable error.

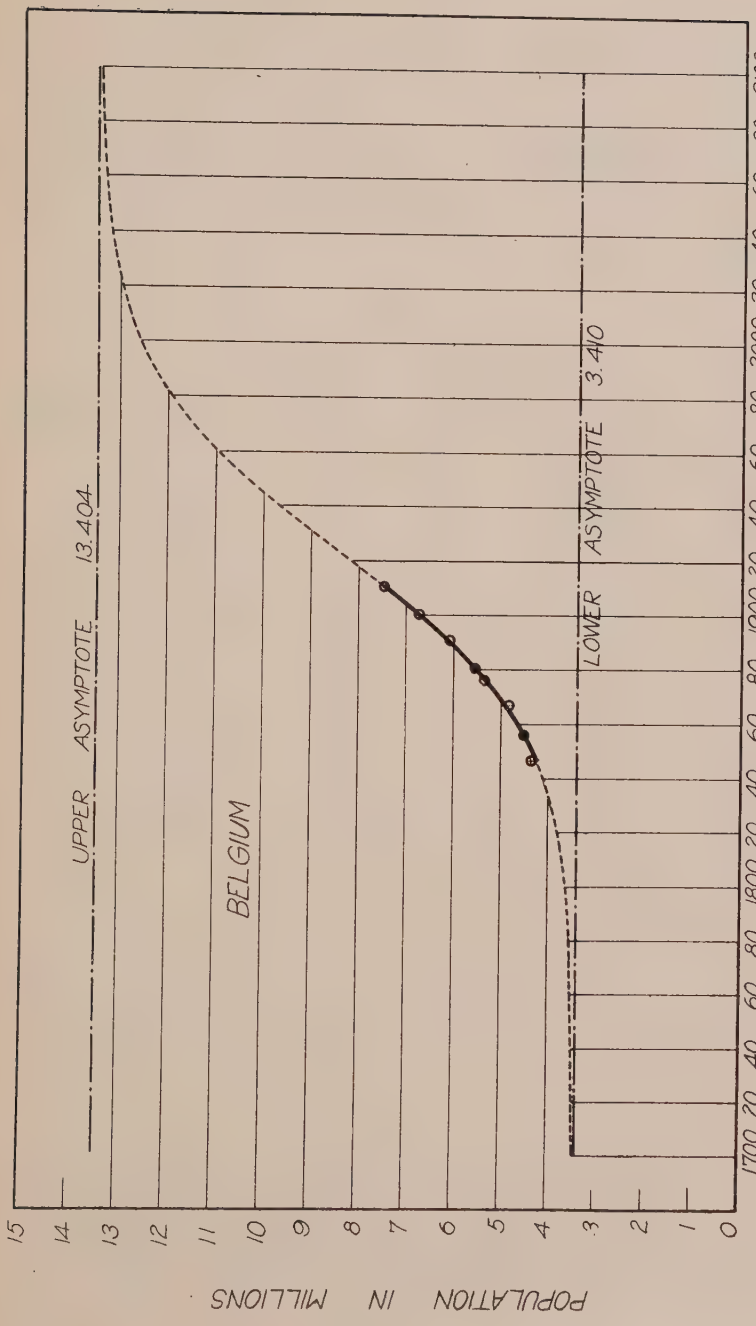

FIG. 106. THE POPULATION GROWTH OF BELGIUM

The population appears to have been in 1910 still in the first half of its current cycle of growth. The indicated point of inflection is early in the year 1932.

The basis of known experience in the case of Denmark is one hundred nine years. The population at 2100 will, according to the curve be within 115,000 people of the estimated upper asymptote. Taking this as sufficiently close we have an extrapolation ratio of 109:190, or nearly 11:19, which is almost as good as the 12:19 of the United States.

TABLE 176

*Denmark**

YEAR	POPULATION IN MILLIONS		YEAR	POPULATION IN MILLIONS	
	Calculated	Observed		Calculated	Observed
Lower asympt.	0.672		1880	1.973	1.970
1700	0.699		1890	2.217	2.170
1720	0.714		1900	2.487	2.450
1740	0.739		1910	2.780	2.780
1760	0.777		1920	3.090	
1780	0.837		1940	3.733	
1800	0.929		1960	4.439	
1801	0.934	0.930	1980	4.885	
1810	0.991		2000	5.312	
1820	1.068		2020	5.629	
1830	1.162		2040	5.853	
1834	1.204	1.220	2060	6.005	
1840	1.275	1.280	2080	6.105	
1850	1.410	1.410	2100	6.171	
1860	1.568	1.600	Upper asympt.	6.286	
1870	1.757	1.780			

* Observed values from Knibbs.

England and Wales

The observed and calculated populations for England and Wales are given in table 177. The curve is:

$$y = 2.373 + \frac{70.670}{1 + 9.719\, e^{-0.0195x}} \qquad \text{(v)}$$

The data are shown graphically in figure 108.

The fit is again obviously excellent. The indicated upper limit of population is approximately 73 million. The recorded figures are all in the first half of the present cycle of growth, the point of inflection having been passed

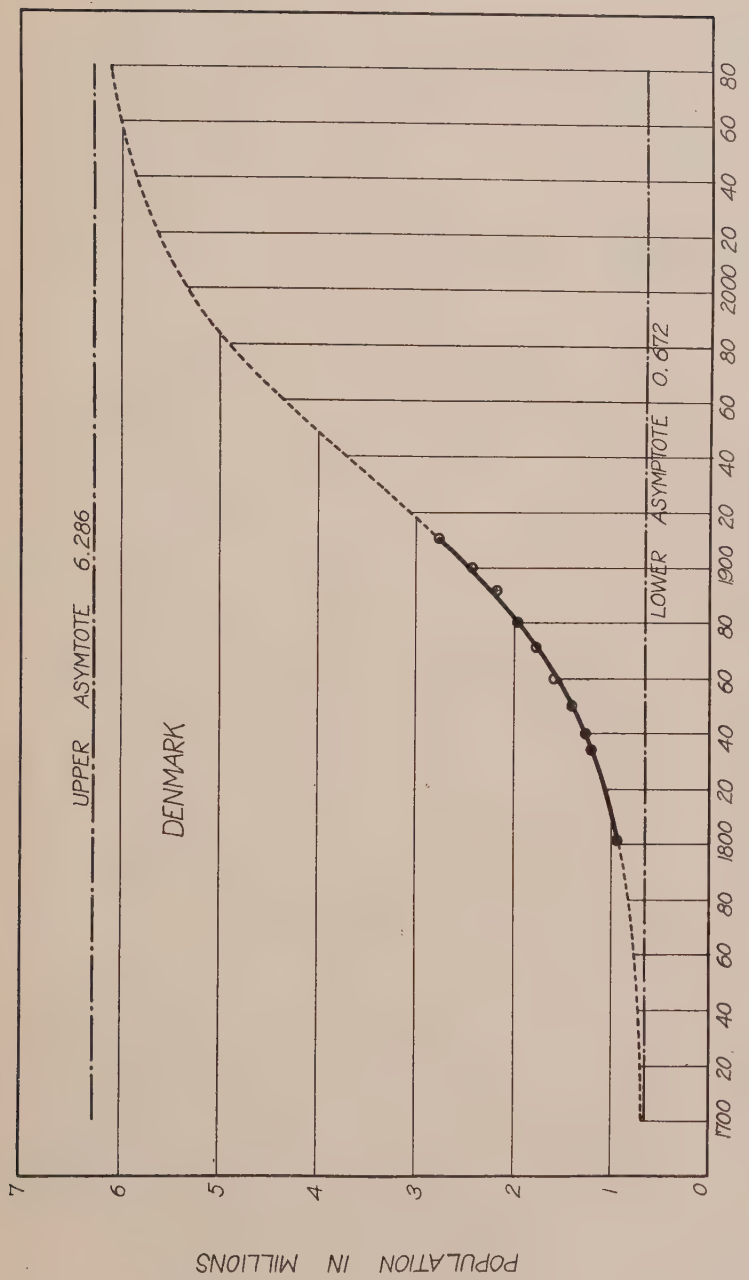

FIG. 107. THE POPULATION GROWTH OF DENMARK

just after the middle of the year 1916. In spite of the decline in the birth rate in England since about 1875 the population has been growing at an ever increasing rate since that time, as already pointed out in Chapter XXIII.

The total basis of known population experience for the calculation of the curve covers one hundred ten years. The estimated population at 2100 is approximately two millions under the predicted asymptotic population. Hence we are extrapolating to get near an asymptote, something over two hundred fifty years on a basis of experience of one hundred ten years.

TABLE 177

*England and Wales**

YEAR	POPULATION IN MILLIONS		YEAR	POPULATION IN MILLIONS	
	Calculated	Observed		Calculated	Observed
Lower asympt.	2.373		1911	35.755	36.070
1700	3.394		1921	39.169	
1720	3.870		1931	42.608	
1740	4.561		1941	45.930	
1760	5.557		1951	49.105	
1780	6.976		1961	52.087	
1801	9.084	8.893	1971	54.842	
1811	10.364	10.164	1981	57.350	
1821	11.853	12.000	1991	59.600	
1831	13.570	13.897	2000	61.404	
1841	15.531	15.914	2020	64.721	
1851	17.746	17.928	2040	67.185	
1861	20.219	20.066	2060	68.967	
1871	22.942	22.712	2080	70.231	
1881	25.894	25.974	2100	71.114	
1891	29.046	29.003	Upper asympt.	73.043	
1901	32.351	32.528			

* Observed data from *Annual Report of the Registrar General.*

The indicated populations prior to the middle of the eighteenth century are probably all too low. At least this is so if we may put any dependence upon Gregory King's[6] estimate of the population of England in 1696, which gives $5\frac{1}{2}$ millions as the probable population of England at that date. As a matter of fact, no one who studies his method of arriving at his estimate can suppose it to have been 2 million or more in excess of the facts. What the case means, we think, is that somewhere between 1600 and 1700 England passed from one cycle of population growth into another, and that as all our

[6] King, G., *Natural and Political Observations and Conclusions upon the State and Condition of England, 1696.*

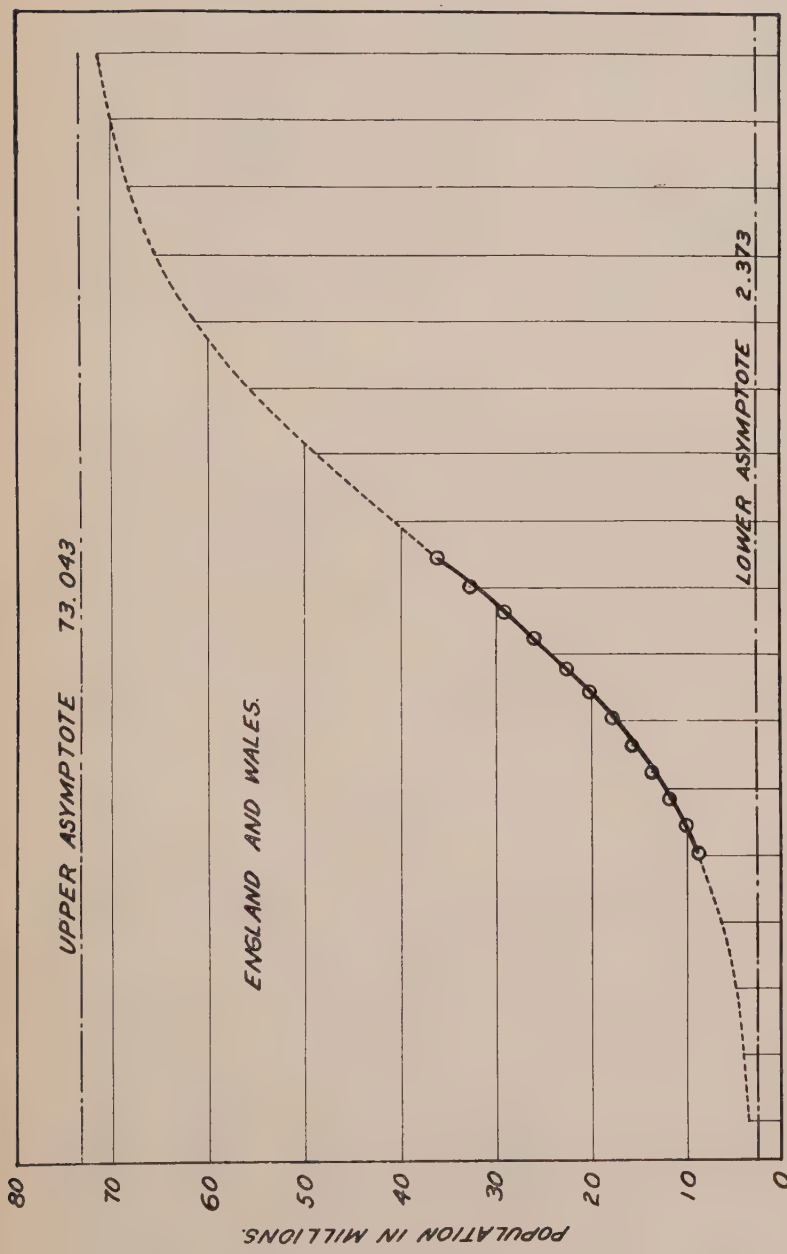

Fig. 108. The Population Growth of England and Wales

observed populations are from a century later on, it is not remarkable that the extrapolation to dates prior to 1700 is considerably in error.

As a predictor of future population Gregory King is not a conspicuous success. He estimates the probable population of England at 1800 as $6\frac{4}{10}\frac{2}{0}$ millions, which was over 2 million in defect; and in 1900 as $7\frac{3}{1}\frac{5}{0}\frac{5}{0}$ millions, which was 25 million in defect. Plainly he had no conception of what industrial development was to do, any more than we have now any adequate notion of what may happen in the next century to upset the predictions of our curves. But it is interesting to note that the shrewd Gregory put the point of inflection of England's population growth not far from 1900, which was not a bad guess!

France

We come now to the consideration of a country whose population growth has excited the interest and attention of virtually all students of any aspect of the population problem. The conditions which have prevailed in France relative to population, in the last quarter of a century at least, have alternately called forth despair or admiration according to whether one's basic social philosophy ran along the "cannon fodder" line or the "high standard of living" line. It was a matter of great interest to us to try the curve on the population figures of this country.

The observed and calculated populations for France appear in table 178, and graphically in figure 109. The equation of the fitted curve is:

$$y = 6.604 + \frac{35.975}{1 + 0.808\, e^{-0.0197x}} \qquad \text{(vi)}$$

A striking difference is at once apparent between this curve and that of any other country so far dealt with. France is near the end of her present cycle of population growth. All the other countries have traversed only half or less of the current cycle. From the standpoint of testing the ability of our curve to describe population growth this is an important difference. Up to this point the evidence has only shown that our curve would describe well the relatively early stages of population growth, where the curve presents a convex face to the base. A curve which dealt very well with this phase of the cycle might fail utterly when put to the task of describing a late phase of the cycle where the observed growth curve presents a concave face to the base. Table 178 and figure 109, taken in conjunction with the evidence already presented, show that our curve suffers no such disability. The known population data for France are very accurately described by the curve,

with the single exception of the year 1872, where theory and observation differ about 600,000, in a total of between 36 and 37 million. There is reason to suppose that the defect in the observed population that year represents the combined effect of three principal causes: one, the Franco-Prussian war itself; the second, the partial occupation of the country by the Germans, and the general disruption of the civil affairs of the country, both consequent upon the war; and finally the loss of Alsace-Lorraine. The effect of each of these causes in reducing the 1872 population count is probably in the inverse order to that in which they are stated above. The effect, however, was obviously only temporary.

TABLE 178

*France**

YEAR	POPULATION IN MILLIONS		YEAR	POPULATION IN MILLIONS	
	Calculated	Observed		Calculated	Observed
Lower asympt.	6.604		1872	36.709	36.103
1500	6.723		1876	37.087	36.906
1550	6.921		1881	37.531	37.672
1600	7.442		1886	37.944	38.219
1650	8.768		1891	38.328	38.343
1700	11.877		1896	38.685	38.518
1720	13.912		1901	39.015	38.962
1740	16.480		1906	39.319	39.252
1760	19.543		1911	39.600	39.602
1780	22.960		1920	40.051	
1801	26.668	26.931	1940	40.836	
1821	30.059	29.871	1960	41.386	
1841	33.063	33.401	1980	41.766	
1861	35.563	35.845	2000	42.027	
1866	36.104	36.495	Upper asympt.	42.579	

* Observed values taken from *Statesman's Year Book.*

The observations cover a period of one hundred and ten years and are at five yearly intervals since 1861, except that the census due in 1871 was taken in 1872. Owing to the fact that France's population is near the end of its current cycle of growth the prediction of the upper asymptote of this cycle has a different order of probable accuracy than any we have so far examined. In the year 2000 the estimated population will be within about 500,000 of the predicted asymptote. So then we are predicting 90 years of growth on one hundred ten years past experience, giving an extrapolation ratio of 11:9. The population in 1910 had less than 3 million to increase in order to reach the predicted asymptote.

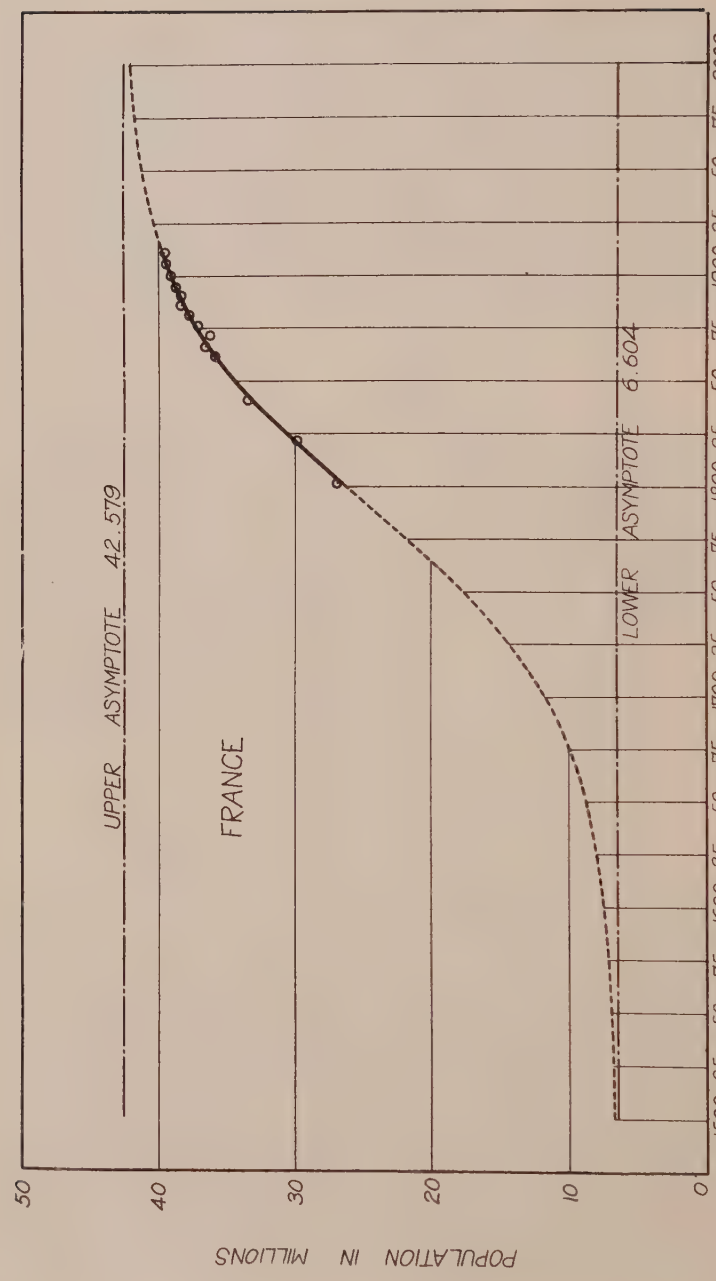

Fig. 109. The Population Growth of France

By the methods outlined in Chapter XXIV we have for France the following probable errors of the constants:

$$k = 35.975 \pm 0.084 \text{ millions of population}$$
$$a_1 = 0.01975 \pm 0.00015$$
$$m = 0.8081 \pm 0.0038$$

These are obviously small errors. Remembering always the proviso "if the basic conditions do not change," we can have a good deal of confidence in the substantial accuracy of the prediction of the upper limit of France's population growth, in the present cycle.

But, in due proportion, our confidence in the estimation of the lower asymptote of the present cycle cannot be great. The indicated lower asymptote is approximately 120,000 lower than the estimated population at 1500. Taking this as a sufficiently close approach to the asymptote we are extrapolating the curve backward over more than three hundred years on an experience of one hundred and ten years, giving an extrapolation ratio of 11:30. Obviously the result will have a larger probable error. One can readily see that a slight alteration or perturbation of the curve over the observed period from 1801 to 1911, might make a large difference in its extrapolated value at dates prior to 1500.

The point of inflection of the theoretical curve is early in the year 1789. Since that time France's population has been growing at an ever decreasing rate.

Germany

The population growth of Germany presented at its outstart a most unpromising problem in curve fitting. All attempts to fit the observations satisfactorily with a single symmetrical growth curve of the form of equation (ix) (Chapter XXIV) failed. A careful examination of the known population data convinced us that we had to deal here with the last part of one cycle of growth (curve of growth concave to base) and the beginning of another cycle (curve of growth convex to the base). Would our curve be able to meet this test of satisfactorily describing these complicated facts?

There were obviously two ways to approach this problem. The first was to fit each assumed cycle separately with a symmetrical growth curve of the sort used in the cases so far considered and then to weld the two together, by assuming that the curve of the new cycle took hold at the point where it was cut by the curve of the old cycle, and carried on by itself thereafter. The second possible approach was to fit the whole series of data as they stood with the generalized form of our curve (equation (xiii) of Chapter

XXIV), and see whether it would in fact, as it theoretically should, take care of two cycles of growth.

Both methods were tried, and the results will be presented here. We may consider first the case in which two symmetrical curves are fitted to the data. The equations are:

$$\text{For the period up to 1855, } y = 10.109 + \frac{34.036}{1+2.495\, e^{-0.0394x}} \qquad \text{(vii)}$$

$$\text{From 1855 on, } \qquad y = 33.587 + \frac{82.944}{1+297.546\, e^{-0.0472x}} \qquad \text{(viii)}$$

The data are presented in table 179, and graphically in figure 110.

It is evident that the graduation is extremely good, considering the peculiar character of the data. What happened in the case of Germany's population is evident. Up to about the time of the Franco-Prussian war Germany had been growing in population along a rather flat drawn out curve, of the sort one would expect for a long settled country quite largely agricultural in its cultural development. The indicated upper asymptote of this cycle of growth was a little over 44 millions.

But already some little time before the Franco-Prussian war Germany was started upon the earliest stages of another cycle of population growth correlated with and dependent upon *industrial* development. After 1870 this industrialization, as every one knows, went on at a very rapid pace, and concurrently population grew at a rate faster than any that had been known for a long time before. Our theory of population growth is able to meet successfully this complication of the overlapping of two cultural cycles.

Considering either of the two curves separately the extrapolation ratio to the time of close approach of the population to the asymptotes is a large one. It is evident that no great dependence can be placed on the absolute figures for the upper asymptote, particularly in view of the known disturbance of the populations consequent upon the late war. It is probable that a graduation of the upper cycle with a 1920 observation included would give a distinctly lower upper asymptote than the one here indicated. The probable error of the indicated lower asymptote of the whole curve must again be large.

It should be understood that in the procedure so far we have fitted the known data as they stood with the two curves (vii) and (viii), and let the asymptote work out to whatever value might be indicated by this process. Suppose now we attempt to fit one single curve of our general type (equation

(xiii), Chapter XXIV) to the whole series of observations assuming now that it starts from a lower asymptote zero.

TABLE 179

*Germany**

YEAR	POPULATION IN MILLIONS		YEAR	POPULATION IN MILLIONS	
	Calculated	Observed		Calculated	Observed
Lower asympt.	10.109		1861	38.281	38.140
1700	10.372		1871	40.867	41 059
1720	10.681		1880	44.230	45 230
1740	11.344		1890	49 430	49 430
1760	12.712		1900	56.370	56.367
1780	15.354		1910	64.930	64.930
1800	19.846		1920	74.518	
1810	22.797		1930	84.165	
1816	24.728	24.833	1940	92.876	
1822	27.725	27.040	1950	100.009	
1831	29.723	29.770	1960	105.394	
1840	32.565	32.790	1970	109.218	
1855	36.586	36.114	1980	111.815	
			1990	113.526	
			2000	114.632	
			Upper asympt.	116.531	

Curve I		Curve II	
YEAR	POPULATION	YEAR	POPULATION
1860	37.681	Lower asympt.	33.587
1870	39.499	1800	33.866
1880	40.867	1830	34.721
1890	41.863	1840	35.392
1900	42.572	1850	36.440
Upper asympt.	44.145		

* Observed figures are from *Statesman's Year Book*.

Calculated figures come from two curves, one running to 1855 and the other from 1855 on.

The equation is:

$$y = \frac{119.552}{1 + e^{1.9416 - 0.0331x + 0.00038x^2 - 0.00000021x^3}} \qquad \text{(ix)}$$

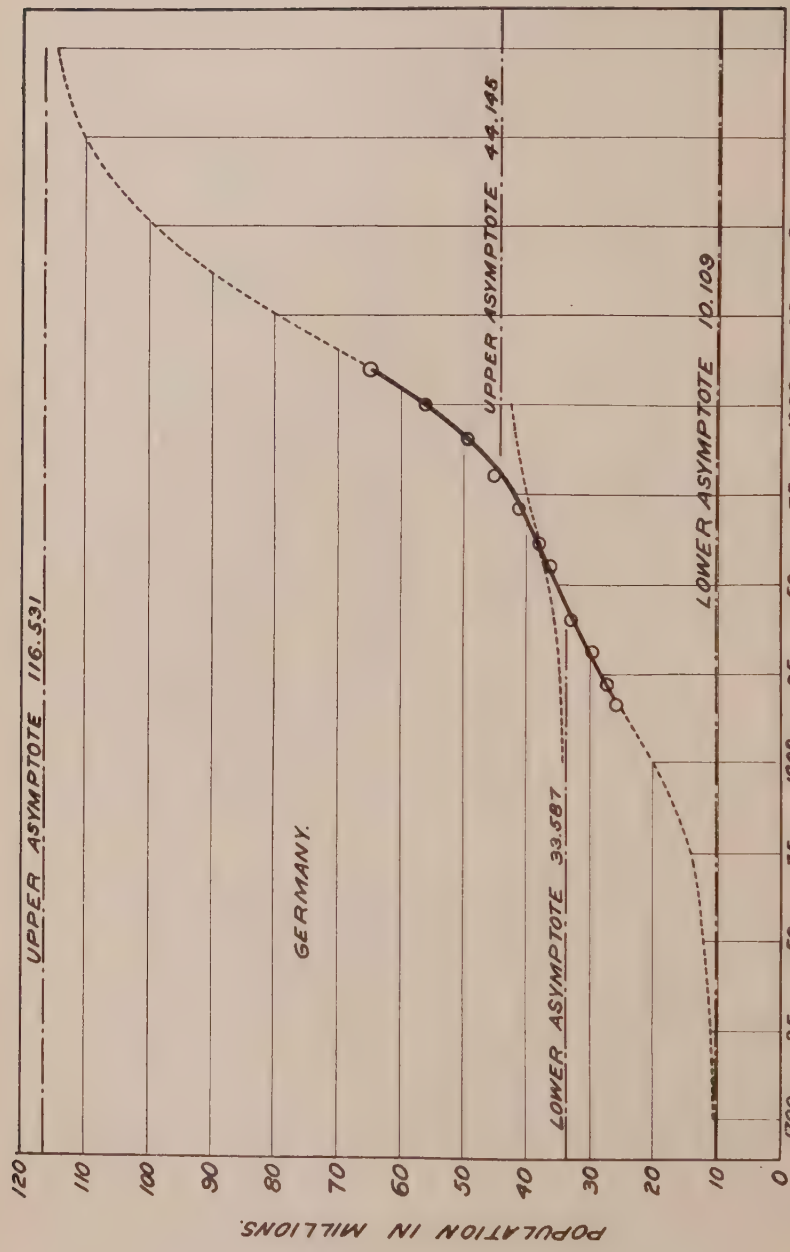

FIG. 110. THE POPULATION GROWTH OF GERMANY

Fitted with two symmetrical growth curves

The comparison of observed and calculated values is given in table 180, and graphically in figure 111.

Obviously the general curve gives quite as good a fit as the two components of figure 110. It demonstrates that the curve has real flexibility and fitting power for the sort of data we are here dealing with. It is furthermore interesting to note that the upper asymptote of population predicted by the curve is only by 3 million different from that predicted by the two component curves. Regarding the lower asymptote it is highly probable that the populations prior to 1780 are more accurately represented by the extrapolation of table 179, than by that of table 180.

TABLE 180

Germany

YEAR	POPULATION IN MILLIONS		YEAR	POPULATION IN MILLIONS	
	Calculated	Observed		Calculated	Observed
Lower asympt.	0.000		1855	36.357	36.114
1700	0.009		1861	37.954	38.140
1720	0.125		1871	41.024	41.059
1740	0.960		1880	44.500	45.230
1760	4.110		1890	50.549	49.430
1780	10.769		1900	56.238	56.367
1790	15.000		1910	64.900	64.930
1800	19.309		1920	75.479	
1816	25.539	24.833	1940	98.592	
1822	27.549	27.040	1960	113.962	
1831	30.237	29.770	1980	118.781	
1840	32.619	32.790	2000	119.499	
1850	35.100		Upper asympt.	119.552	

Altogether the case of Germany shows that our population curve is capable of meeting successfully a complicated phenomenal situation in growth. This case greatly strengthens the idea that we have here a *real* descriptive law of growth.

Hungary

For Hungary the known data are meager, and the whole case merits only brief treatment. The equation to the fitted curve is:

$$y = 11.326 + \frac{27.673}{1 + 65.805\, e^{-0.0324x}} \qquad (x)$$

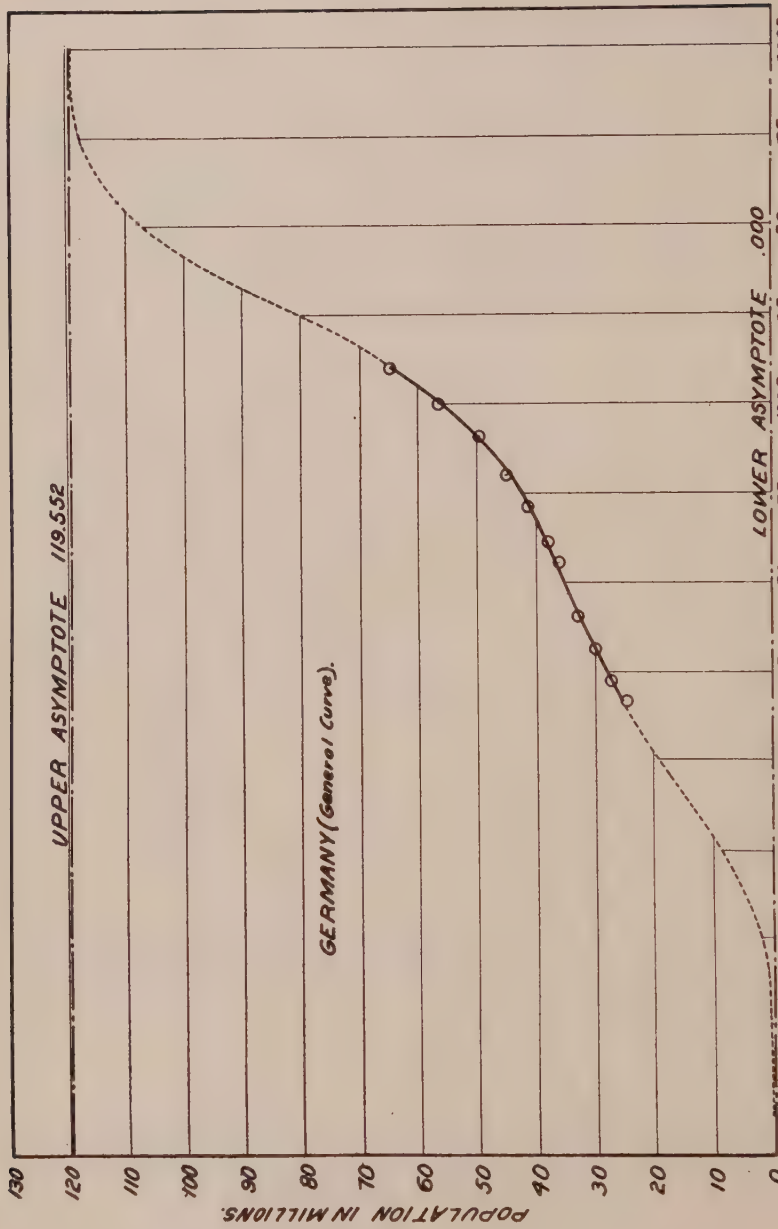

FIG. 111. THE POPULATION GROWTH OF GERMANY
Fitted with the general curve for population growth

The data are given in table 181, and graphically in figure 112.

While the fit is good this is not remarkable, since we are striking a four constant curve through only 6 points. It ought to fit. Owing to the meagerness of the observations no reliance could possibly be placed upon the extrapolation beyond a short distance from the observed range. The indicated point of inflection of this theoretical curve is early in the year 1929. But of course since the war Hungary no longer exists as a political entity as it did before. Hence the extrapolation to future populations has no real meaning.

TABLE 181

*Hungary**

YEAR	POPULATION IN MILLIONS		YEAR	POPULATION IN MILLIONS	
	Calculated	Observed		Calculated	Observed
Lower asympt.	11.326		1910	20.997	20.886
1700	11.342		1920	23.122	
1720	11.357		1930	25.349	
1740	11.386		1940	27.569	
1760	11.441		1950	29.664	
1780	11.544		1960	31.552	
1800	11.740		1970	33.181	
1810	11.896		1980	34.532	
1820	12.107		1990	35.617	
1830	12.395		2000	36.468	
1840	12.783		2020	37.615	
1850	13.301		2040	38.257	
1857	13.759	13.769	2060	38.606	
1869	14.772	15.417	2080	38.792	
1880	15.999	15.739	2100	38.890	
1890	17.396	17.464	Upper asympt.	38.999	
1900	19.069	19.255			

* Observed population figures taken from *Statesman's Year Book.*

Italy

The observed figures for the population growth of Italy cover a much longer period than those for Hungary, but the earlier ones are estimates based upon partial counts only.

The equation to the fitted curve is:

$$y = 11.561 + \frac{37.495}{1 + 5.987\, e^{-0.0201x}} \qquad \text{(xi)}$$

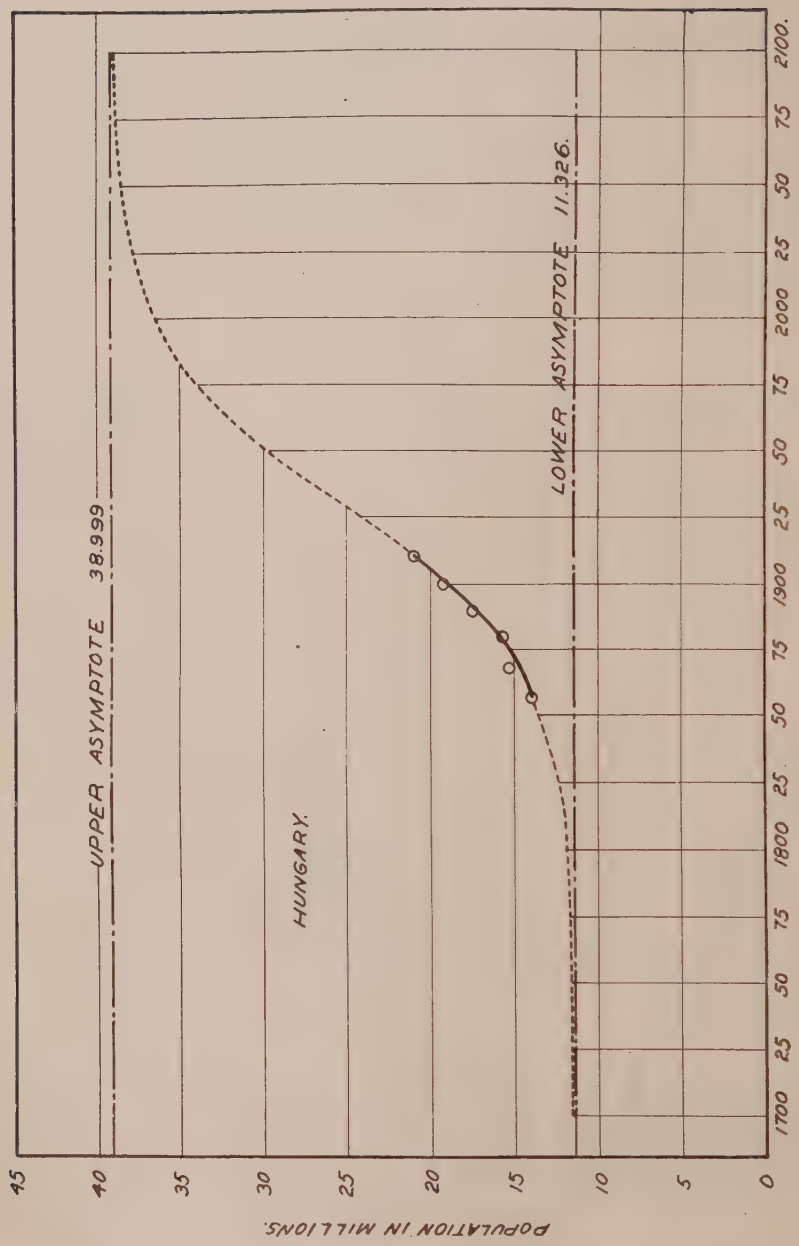

Fig. 112. The Population Growth of Hungary

The data are given in table 182, and graphically in figure 113.

The theoretical curve is seen to fit very accurately the 10 observed points. The indicated lower asymptote of the present cycle of growth was within practically 100,000 of the indicated population in 1600. The extrapolation ratio to 1600 is therefore approximately 14:17, a fairly high value relative to some of the other countries. In the other direction it has a smaller value, and again the area of the country has been altered as a result of the war. The indicated point of inflection in Italy's present cycle of growth occurred at the beginning of the year 1889.

TABLE 182

*Italy**

YEAR	POPULATION IN MILLIONS		YEAR	POPULATION IN MILLIONS	
	Calculated	Observed		Calculated	Observed
Lower asympt.	11.561		1861	25.172	25.017
1600	11.673		1871	26.969	26.801
1620	11.728		1881	28.812	28.460
1640	11.810		1891	30.695	
1660	11.932		1901	32.569	32.475
1680	12.113		1911	34.399	34.671
1700	12.380		1920	35.980	
1720	12.773		1940	39.169	
1740	13.345		1960	41.812	
1760	14.167		1980	43.880	
1770	14.700	14.689	2000	45.429	
1780	15.329		2020	46.550	
1800	16.938	17.237	2040	47.342	
1816	18.584	18.383	2060	47.892	
1820	19.055		2080	48.270	
1825	19.676	19.727	2100	48.526	
1840	21.757		Upper asympt.	49.056	
1848	22.995	23.617			

* First census 1871. Earlier counts are estimates based on partial counts. Figures from *Encyclopedia Britannica*, checked against figures in *Statesman's Year Book*.

Norway

In the case of Norway there are available eleven observed points extending from 1800 to 1920 inclusive. The equation of the theoretical curve is:

$$y = 0.534 + \frac{3.274}{1 + 8.595\, e^{-0.0224x}} \qquad \text{(xii)}$$

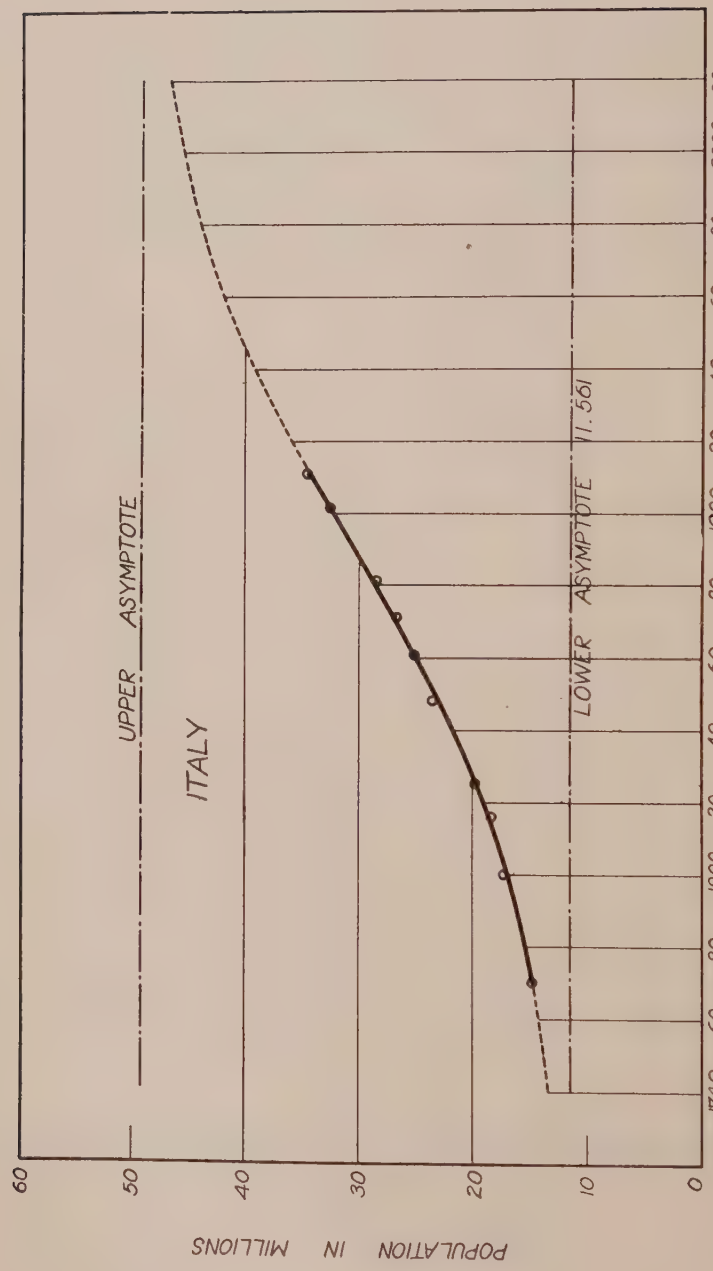

Fig. 113. The Population Growth of Italy

The point of inflection of this theoretical curve was at the beginning of the year 1896. Since that time Norway's population has been growing at a decreasing rate.

The data are given in table 183, and graphically in figure 114.

The case calls for no special comment. The graduation is obviously excellent. The extrapolation ratio to a point within 40,000 of the lower asymptotic population is 1.2, a value high enough to give some fair degree of confidence in the result. The indicated upper asymptote of the present cycle of Norway's growth is a population of $3\frac{4}{5}$ millions.

TABLE 183

Norway

YEAR	POPULATION IN MILLIONS		YEAR	POPULATION IN MILLIONS	
	Calculated	Observed		Calculated	Observed
Lower asympt.	0.534		1890	2.061	1.990
1700	0.574		1900	2.244	2.220
1720	0.596		1910	2.425	2.390
1740	0.630		1920	2.600	2.646
1760	0.682		1930	2.766	
1780	0.761		1940	2.918	
1800	0.875	0.880	1960	3.177	
1810	0.950		1980	3.375	
1820	1.038		2000	3.517	
1825	1.088	1.050	2020	3.616	
1835	1.199	1.190	2040	3.683	
1845	1.326	1.330	2060	3.727	
1855	1.468	1.490	2080	3.756	
1865	1.624	1.700	2100	3.774	
1875	1.793	1.820	Upper asympt.	3.808	
1880	1.880				

Scotland

Scotland's recorded population history furnishes 13 observed points. The equation to the fitted curve is:

$$y = 0.178 + \frac{8.063}{1 + 4.669\, e^{-0.0164x}} \qquad \text{(xiii)}$$

The present cycle has passed its point of most rapid growth, inflection having occurred in the first quarter of the year 1894. The observations in general occupy about the middle fifth of the whole present cycle, with the result that both asymptotes must have fairly large probable errors relatively.

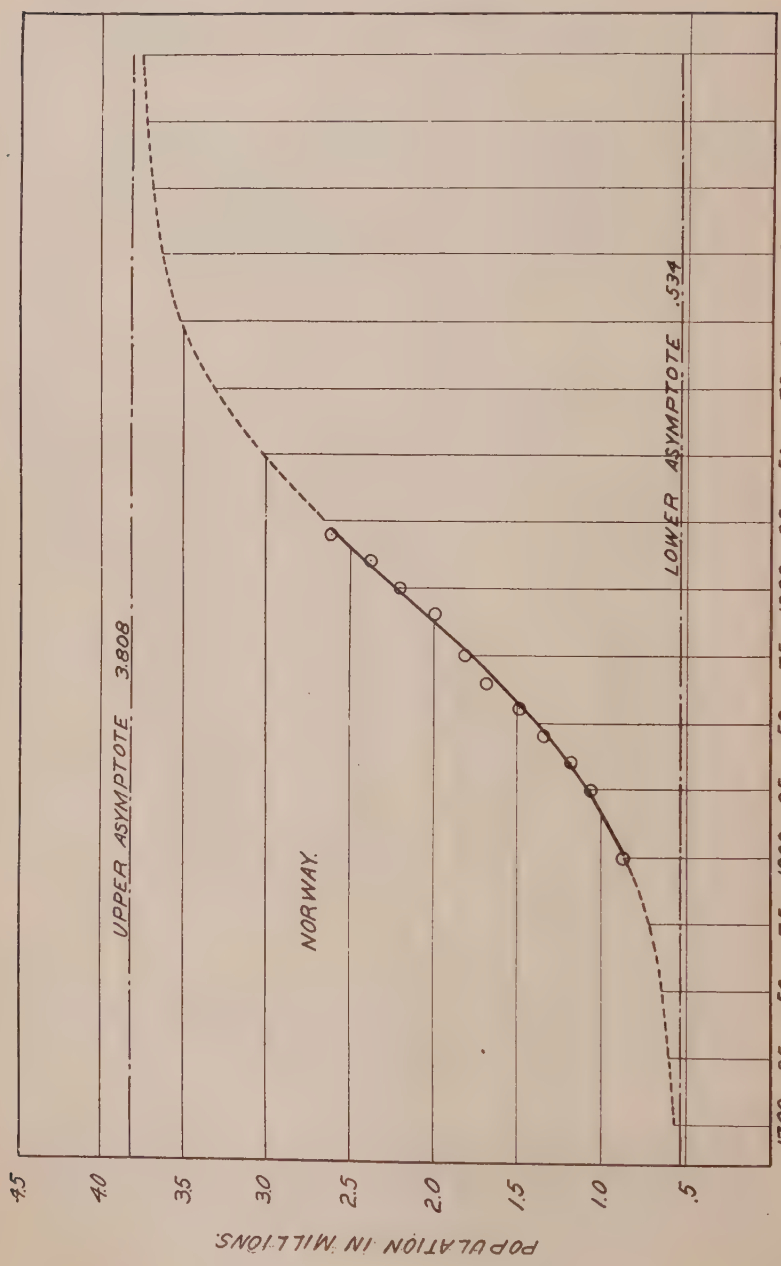

Fig. 114. The Population Growth of Norway

The observed and calculated values are presented in table 184, and graphically in figure 115.

The fit is obviously well-nigh perfect. The predicted asymptotes are probably somewhat too low in the case of the lower, and too high in the case of the upper.

TABLE 184

*Scotland**

YEAR	POPULATION IN MILLIONS		YEAR	POPULATION IN MILLIONS	
	Calculated	Observed		Calculated	Observed
Lower asympt.	0.178		1871	3.454	3.360
1600	0.243		1881	3.777	3.736
1650	0.322		1891	4.106	4.026
1700	0.500		1901	4.434	4.472
1720	0.618		1911	4.761	4.761
1740	0.776		1921	5.080	4.883
1760	0.984		1940	5.653	
1780	1.255		1960	6.191	
1800	1.600		1980	6.650	
1801	1.619	1.608	2000	7.028	
1811	1.823	1.806	2020	7.327	
1821	2.047	2.092	2040	7.561	
1831	2.293	2.364	2060	7.738	
1841	2.558	2.620	2080	7.873	
1851	2.842	2.889	2100	7.972	
1861	3.141	3.062	Upper asympt.	8.240	

* Observed values are census counts as given in *Statesman's Year Book*.

Servia

Servia is taken as an example of a small country, with a civilization and racial background in some respects quite different from any so far discussed. For such a country it also has a fairly long recorded population history.

The equation to the fitted curve is:

$$y = 0.148 + \frac{3.905}{1 + 50.282\, e^{-0.0435x}} \qquad \text{(xiv)}$$

The data are given in table 185, and figure 116. The indicated time of most rapid growth of Servia's population was at the beginning of 1890. As in some of the other cases the extrapolation of future populations has no significance now, because of the alteration of boundaries by the war, or loss of identity as a separate political unit.

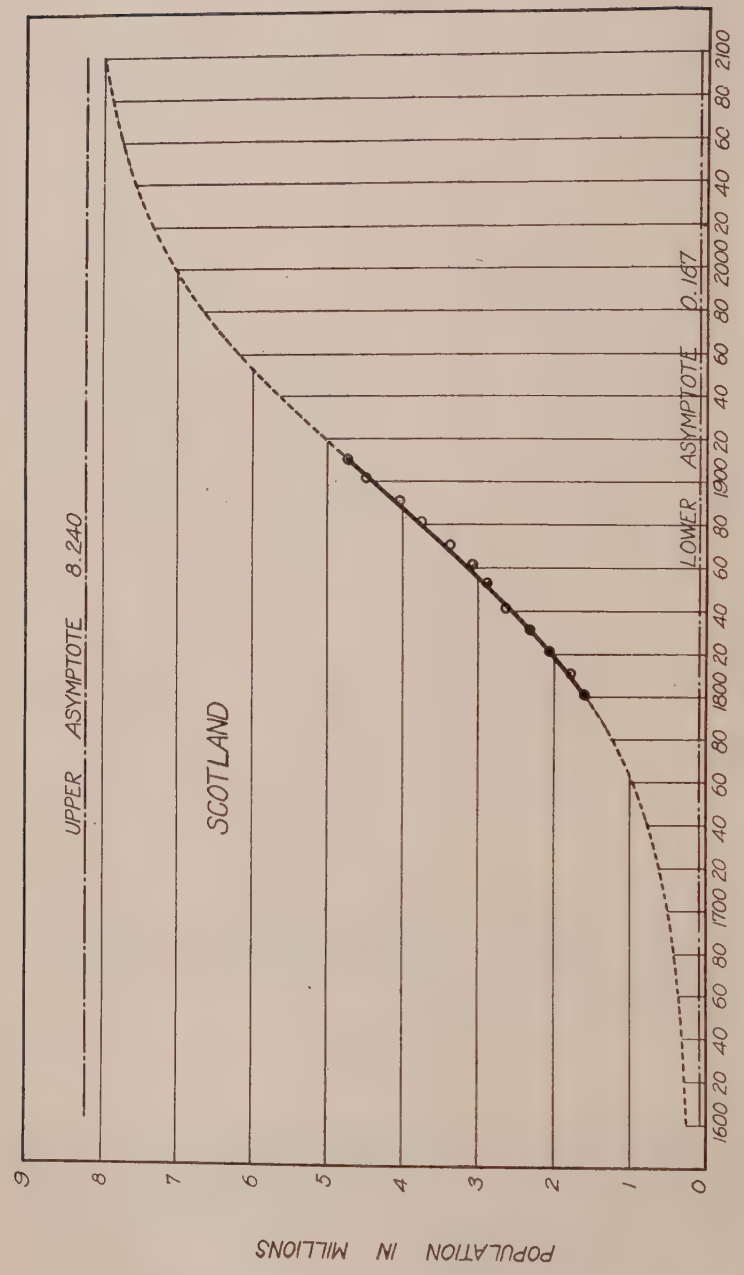

FIG. 115. THE POPULATION GROWTH OF SCOTLAND

The curve fits the observed data very well, and probably the extrapolated values towards the lower asymptote are reasonably near to the facts.

Sweden

The case of Sweden is of particular interest in the present connection because of its long and accurate recorded population history. We have 18 observed points, and such a long array tests the power of the curve to deal with population data.

The equation to the fitted curve is:

$$y = 1.535 + \frac{6\,336}{1 + 7.265\, e^{-0.0230x}} \qquad \text{(xv)}$$

TABLE 185

*Servia**

YEAR	POPULATION IN MILLIONS		YEAR	POPULATION IN MILLIONS	
	Calculated	Observed		Calculated	Observed
Lower asympt.	0.148		1884	1.851	1.900
1700	0.149		1890	2.100	2.162
1720	0.150		1895	2.312	2.312
1740	0.154		1900	2.519	2.493
1760	0.162		1905	2.716	2.689
1780	0.180		1910	2.900	2.912
1800	0.224		1920	3.220	
1820	0.325		1940	3.655	
1830	0.415	0.400	1960	3.876	
1840	0.546		1980	3.977	
1860	0.980	1.000	2000	4.021	
1872	1.372	1.319	Upper asympt.	4.053	
1874	1.447	1.350			

* Observed values taken from Knibbs' *Mathematical Theory of Population.*

The observed and calculated populations are shown in table 186, and figure 117.

The curve indicates that Sweden had her point of most rapid population growth in the present cycle nearly forty years ago; to be exact in the first quarter of the year 1886. Since then her rate of increase has been falling off.

The test imposed in fitting a four constant curve to 18 observed points is beautifully met. In the long period from 1750 to 1920 inclusive our curve describes the actual population growth of Sweden with great accuracy.

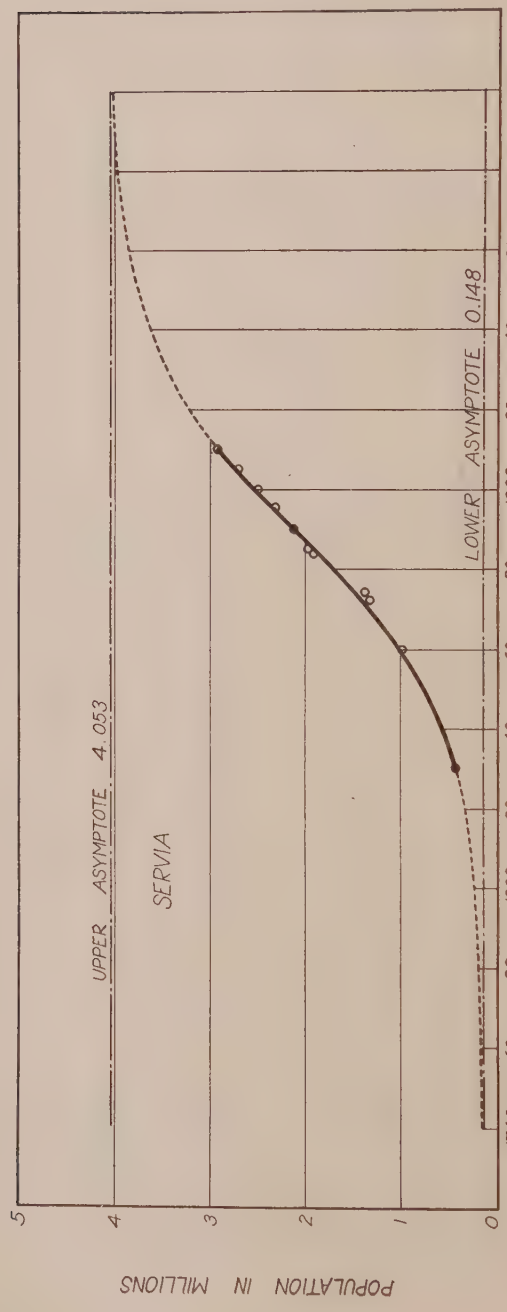

FIG. 116. THE POPULATION GROWTH OF SERVIA

The probable errors of the constants are:

$$k = 6.336 \pm 0.054 \text{ millions of population}$$
$$a_1 = 0.023006 \pm 0.000019$$
$$m = 7.265 \pm 0.016$$

With this case we may leave the consideration of European countries for the present. It has been shown that the curve is able satisfactorily to deal with the growth of the most diverse sorts of populations and cultures, ranging all the way from generally agricultural to some of the most highly industrialized populations yet known.

TABLE 186

Sweden

YEAR	POPULATION IN MILLIONS		YEAR	POPULATION IN MILLIONS	
	Calculated	Observed		Calculated	Observed
Lower asympt.	1.535		1870	4.119	4.168
1700	1.621		1880	4.477	4.566
1720	1.670		1890	4.841	4.785
1740	1.747		1900	5.202	5.136
1750	1.800	1.763	1910	5.549	5.522
1760	1.864	1.893	1920	5.876	5.904
1770	1.944	2.030	1940	6.446	
1780	2.041	2.118	1960	6.890	
1790	2.160	2.158	1980	7.215	
1800	2.302	2.347	2000	7.440	
1810	2.471	2.378	2020	7.592	
1820	2.669	2.585	2040	7.692	
1830	2.900	2.888	2060	7.757	
1840	3.162	3.139	2080	7.798	
1850	3.455	3.483	2100	7.825	
1860	3.776	3.860	Upper asympt.	7.871	

THE POPULATION GROWTH OF SOME ASIATIC COUNTRIES

Japan

Japan furnished an interesting problem in the discussion of population growth for the reason that a sharp transition from one type of culture to another occurred in comparatively recent times. Furthermore we are able to get some reasonably reliable estimates of the size of the population towards the end of the earlier epoch. We have accordingly thought it desirable to treat the material in two ways: first to fit the single cycle, symmetrical curve (equation (ix) of Chapter XXIV) just as has been done with the other countries. This procedure will give no information about

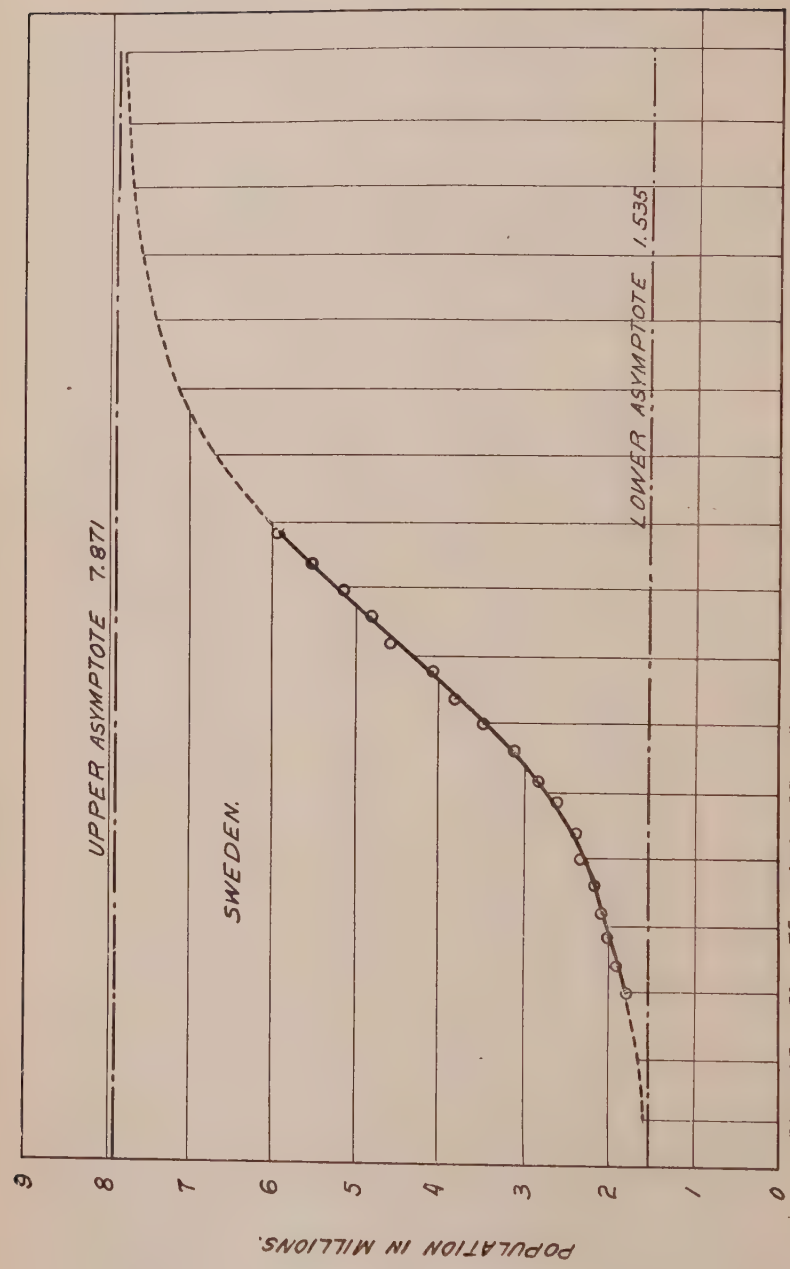

Fig. 117. The Population Growth of Sweden

the prior cycle of growth except an approximation to the population size attained at its upper asymptote. A second course was to fit the data with the generalized curve (equation (xiii) of Chapter XXIV) and see what indication it gave regarding the prior cycle, and in general how it would fit the data.

Proceeding on the first line we have the equation

$$y = 25.000 + \frac{64.271}{1 + 88.982\, e^{-0.0368x}} \qquad \text{(xvi)}$$

The observed population, and the ordinates of equation (xvi) are shown in table 187, and graphically in figure 118. The indicated time of most

TABLE 187

*Japan**

YEAR	POPULATION IN MILLIONS		YEAR	POPULATION IN MILLIONS	
	Calculated	Observed		Calculated	Observed
Lower asympt.	25.0		1910	50.2	50.5
1700	25.0		1920	56.0	56.0
1732	25.1	25.5	1940	67.4	
1760	25.2		1960	76.5	
1800	25.7	26.1	1980	82.5	
1820	26.5	25.9	2000	85.8	
1840	28.0		2020	87.6	
1860	31.0		2040	88.4	
1880	36.3		2060	88.9	
1882	37.0	36.7	2080	89.1	
1890	40.1	40.4	2100	89.2	
1900	44.8	44.8	Upper asympt.	89.3	

* The three early population counts are the results of religious censuses. The later counts are as given in *Encyclopedia Britannica.*

rapid growth is at the beginning of the year 1920, a value which seems entirely reasonable.

The graduation is on the whole good. The curve goes a little under the 1800 observation, and a little over the 1820 one. But both these are estimates and we should pay no attention to the discrepancies were it not for the fact that it is known that Japan was at about this time passing from one kind of culture to another. The indicated upper asymptote from this curve is 89 $\frac{3}{10}$ millions, to be closely approached about two centuries hence.

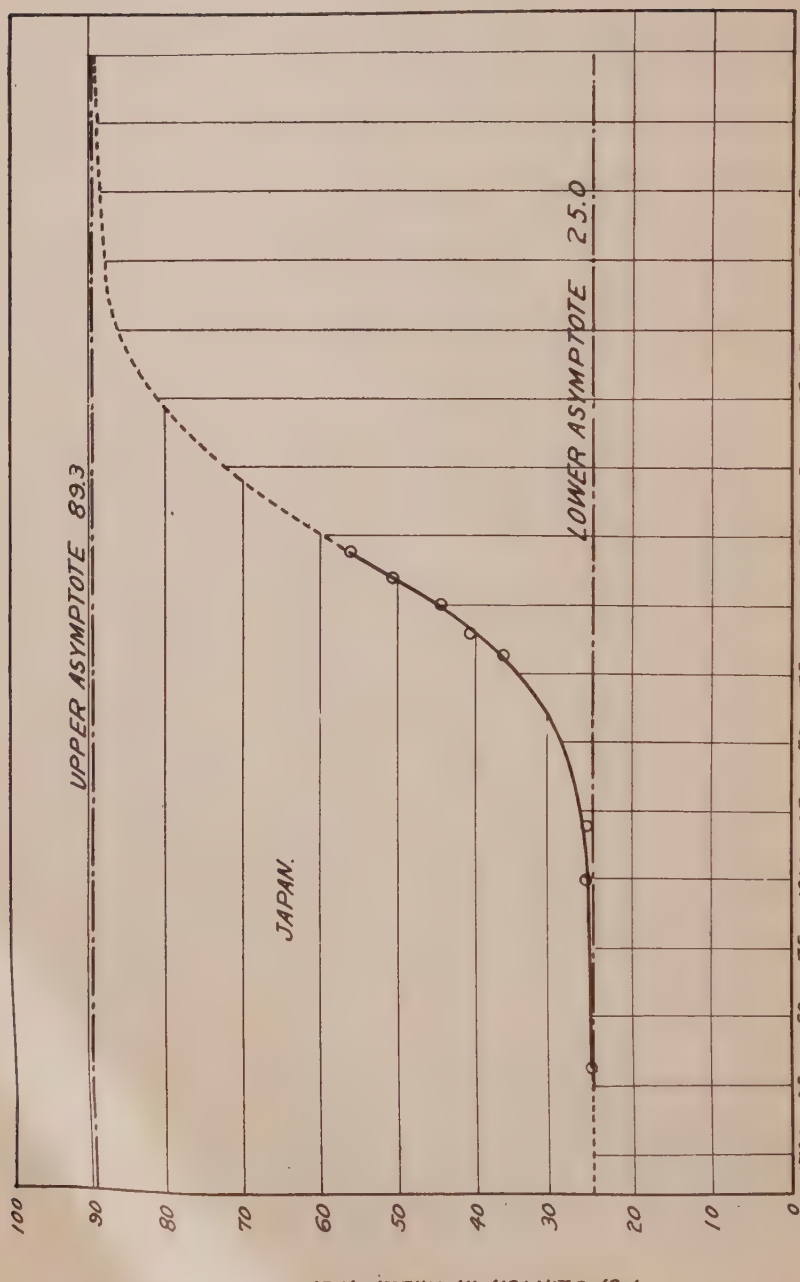

FIG. 118. THE POPULATION GROWTH OF JAPAN
Fitted with single, symmetrical growth curve

The generalized curve, to which we may now turn, has the equation, in this case:

$$y = \frac{86.641}{1 + e^{0.8765 - 0.000435x - 0.000043x^2 - 0.00000047x^3}} \qquad \text{(xvii)}$$

This starts from a lower asymptote of zero, and the population is estimated to have reached a magnitude of 661,000 in 1560.

The comparison of this curve with the observations is given in table 188, and figure 119.

This curve gives a somewhat better fit to the observed points than the other simpler one. But it must be remembered that we are fitting 8 obser-

TABLE 188

Japan (General Equation)

YEAR	POPULATION IN MILLIONS		YEAR	POPULATION IN MILLIONS	
	Calculated	Observed		Calculated	Observed
Lower asympt.	0.000		1820	26.004	25.900
1560	0.661		1840	27.597	
1580	1.812		1860	30.795	
1600	4.242		1870	33.201	
1620	8.071		1882	36.967	36.700
1640	12.809		1890	40.072	40.450
1660	17.530		1900	44.662	44.830
1680	21.413		1910	49.988	50.500
1700	24.066		1920	55.902	55.960
1720	25.500		1940	68.126	
1732	25.876	25.500	1960	77.983	
1740	25.972		1980	83.578	
1760	25.854		2000	85.820	
1780	25.551		2020	86.474	
1800	25.465	26.100	Upper asympt.	86.641	

vations with a 5 constant curve, and naturally expect to get good results if the curve used has any aptitude at all for the case in hand. This case, taken in connection with the results on Germany's population growth, is however impressive in its indication of the power of the general curve to deal with complex growth results.

It is interesting to note that the final upper asymptote indicated by the general curve is $86\frac{3}{5}$ millions of population. This figure is less than 3 millions different from that given by the simple symmetrical curve first tried.

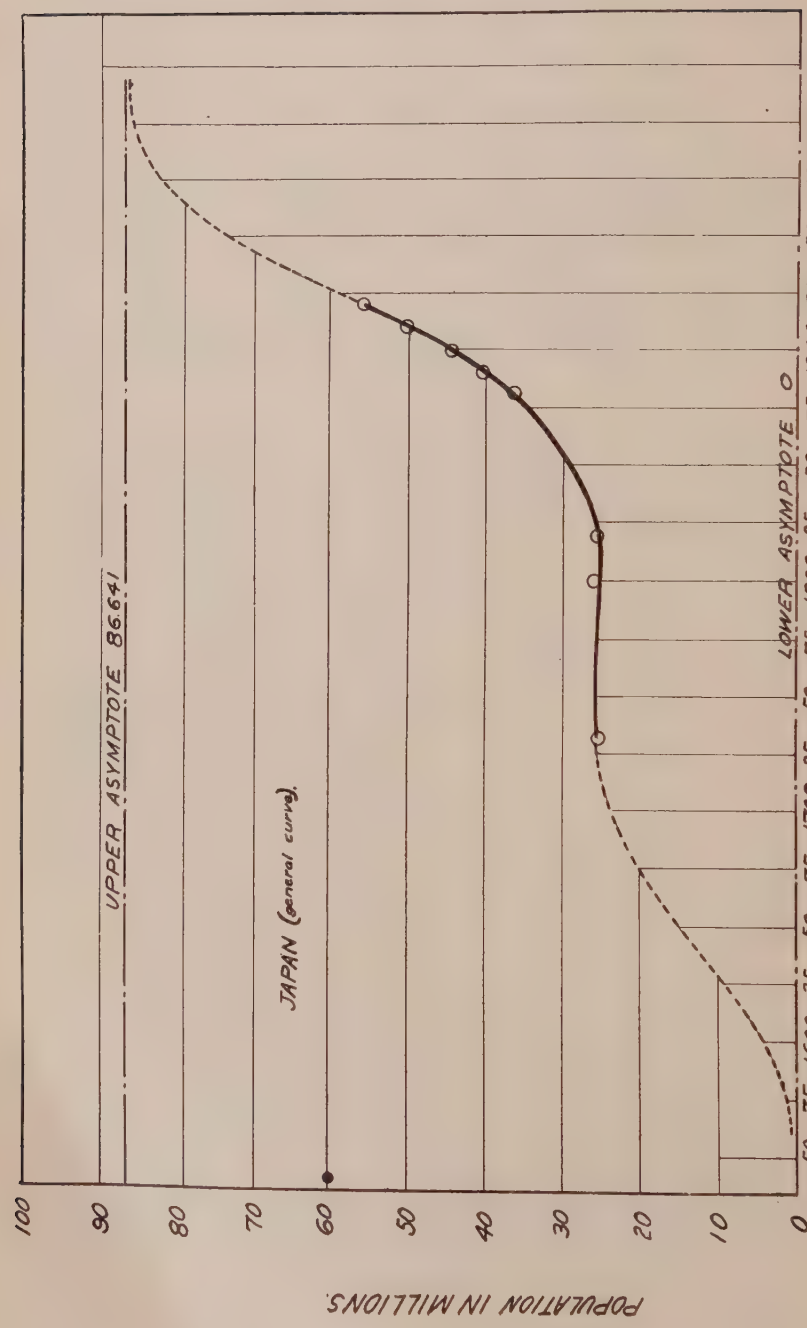

Fig. 119. The Population Growth of Japan
Fitted with general curve

Java

For Java, representing a wholly different sort of culture than any group yet dealt with, we have 9 observations (or estimates) of population, extending from 1780 to 1920 inclusive. The equation of the fitted curve is:

$$y = 1.572 + \frac{49.140}{1 + 28.847\, e^{-0.0356x}} \qquad \text{(xviii)}$$

The indicated point of inflection of Java's population growth was about April, 1894.

TABLE 189

*Java**

YEAR	POPULATION IN MILLIONS		YEAR	POPULATION IN MILLIONS	
	Calculated	Observed		Calculated	Observed
Lower asympt.	1.57		1880	19.99	19.79
1700	1.62		1890	24.24	23.91
1720	1.67		1900	28.61	28.75
1740	1.77		1905	30.75	30.10
1760	1.98		1910	32.82	
1780	2.39	2.02	1920	36.65	35.02
1790	2.74		1930	39.94	
1800	3.22		1940	42.64	
1810	3.89	3.77	1950	44.77	
1820	4.82		1960	46.39	
1830	6.08		1970	47.61	
1840	7.76		1980	48.49	
1845	8.79	9.54	1990	49.14	
1850	9.96		2000	49.60	
1860	12.74		Upper asympt.	50.71	
1870	16.11	16.38			

* Observed values taken from the reports of the Philippine census for 1903, and from *Statesman's Year Book.*

The observed and calculated populations are shown in table 189, and figure 120.

The observations cover a good part of the present cycle of growth, and the agreement between observations and theory is excellent. The extrapolated populations seem reasonable.

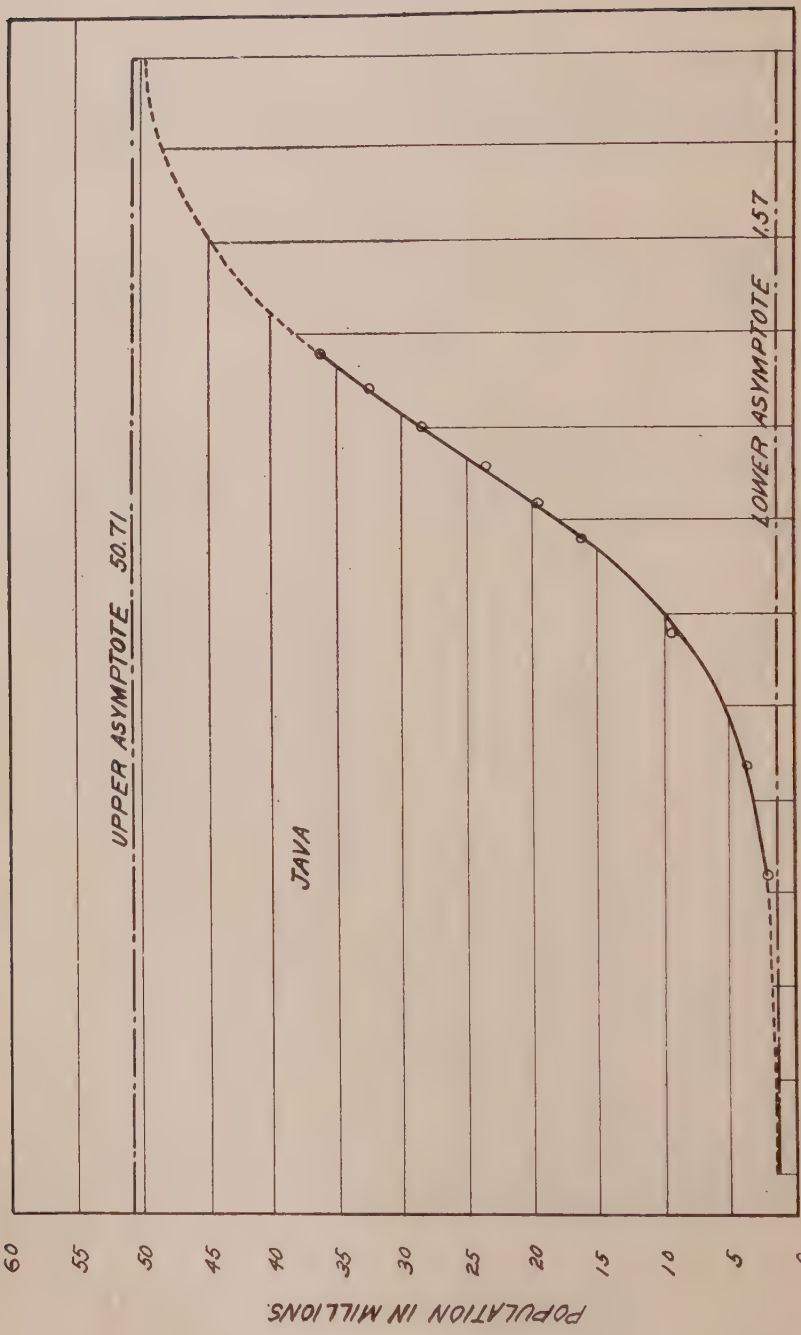

Fig. 120. The Population Growth of Java

Philippine Islands

There are population records for the Philippine Islands, exclusive of wild tribes, from 1800 to 1903 inclusive. The equation for the fitted curve is:

$$y = 0.285 + \frac{10.060}{1 + 6.951\, e^{-0.0255x}} \qquad \text{(xix)}$$

The calculated time of most rapid growth of the population was early in the year 1876.

TABLE 190

*Philippine Islands**

YEAR	POPULATION IN MILLIONS		YEAR	POPULATION IN MILLIONS	
	Calculated	Observed		Calculated	Observed
Lower asympt.	0.285		1890	6.200	
1700	0.397		1894	6.446	6.491
1720	0.470		1900	6.804	
1740	0.589		1903	6.978	6.988
1760	0.781		1910	7.365	
1780	1.085		1920	7.875	
1800	1.550	1.561	1930	8.315	
1810	1.860		1940	8.697	
1812	1.929	1.933	1950	9.019	
1820	2.229		1960	9.286	
1829	2.614	2.593	1970	9.504	
1830	2.660		1980	9.679	
1840	3.153	3.096	1990	9.823	
1850	3.703	3.857	2000	9.935	
1858	4.177	4.290	2020	10.095	
1860	4.300		2040	10.193	
1870	4.927	4.712	2060	10.253	
1879	5.504	5.487	2080	10.290	
1880	5.568		2100	10.312	
1885	5.886	5.839	Upper asympt.	10.345	

* Observed values are exclusive of wild tribes. The figures are taken from the 1903 Census of the Philippine Islands.

The observations and fitted curve are shown in table 190, and figure 121. The agreement between the curve and the observed population is extremely close. A better fit could not be expected. The asymptotic populations doubtless have fairly large probable errors, but they appear in no way unreasonable, judged from the known facts.

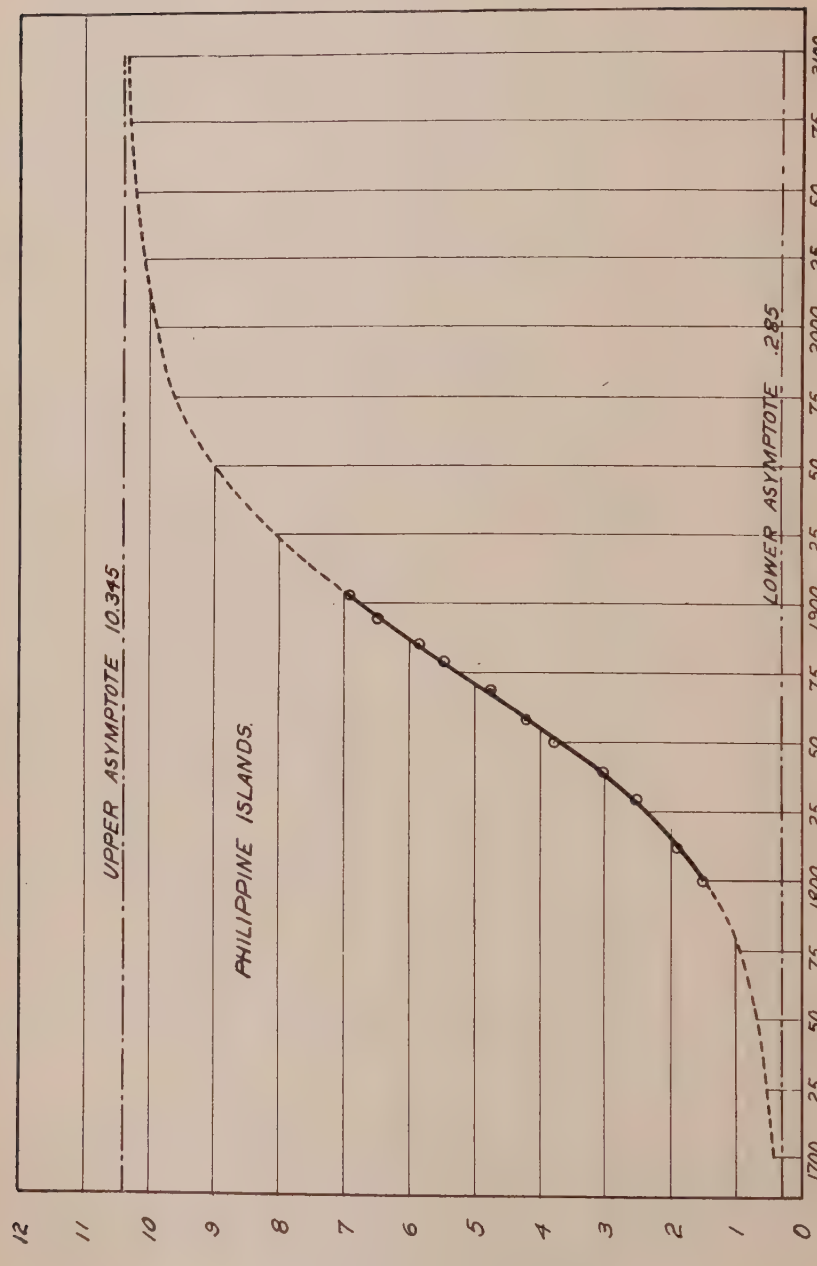

Fig. 121. The Population Growth of the Philippine Islands

THE GROWTH OF THE POPULATION OF THE WORLD

The population of the whole world has never been counted. Doubtless it will be a long time before it is. But a great many estimates have been

TABLE 191

Estimates of world's population (from Knibbs)

YEAR	AUTHORITY	ESTIMATE (MILLIONS)	YEAR	AUTHORITY	ESTIMATE (MILLIONS)
1660	Riccioli	1,000	1840	v. Roon	864
1685	Isaak Vossius	500	1843	Balbi	739
1740	Nic. Struyck	500	1843	H. Berghaus	1,272
1672	Riccioli	1,000	1845	Michelot	1,009
1742	J. P. Sussmilch	950 to 1,000	1854	v. Reden	1,135
1753	Voltaire	1,600	1889	Dieterici	1,288
1761	J. P. Sussmilch	1,080	1866	E. Behm	1,350
1789	W. Black	800 to 1,000	1868	Kolb	1,270
1804	Malte-Brun	640	1868	E. Behm	1,375
1804	Volney	437	1870	E. Behm	1,359
1805	Pinkerton	700	1872	Behm and Wagner	1,377
1805	Fabri	700	1873	Behm and Wagner	1,391
1809	G. Hassel	682	1874	Behm and Wagner	1,391
1810	*Almanach de Gotha*	682	1878	Levasseur	1,439
1812	Morse	766			
1813	Graberg v. Hemso	686	1880	Behm and Wagner	1,456
1816	A. Balbi	704	1882	Behm and Wagner	1,434
1822	Reichard	732	1883	Behm and Wagner	1,433
1824	G. Hassel	938	1886	Levasseur	1,483
1828	G. Hassel	850	1891	Ravenstein	1,467
1828	I. Bergius	893	1896	*Statesman's Year Book*	1,493
1828	A. Balbi	737	1903	Juraschek	1,512
1828	Balbi	847	1906	Juraschek	1,538
1833	Stein	872	1910	*Annuaire Statistique d.*	
1838	Franzl	950		*l. Rep. Francaise*	
1838	V. Rougemont	850		Juraschek	1,610
1840	Omalius d'Halloy	750	1913	Knibbs	1,632
1840	Bernouli	764	1914	Knibbs	1,649

made of world population. The more significant of these have been collected together by Knibbs[7] in a table here reproduced as table 191.

It seemed of interest to see what our population curve would do when applied to these estimates of world population. From Knibbs's table given

[7] Knibbs, G. H., The mathematical theory of population, of its character and fluctuations, and of the factors which influence them; *Census of Commonwealth of Australia*, Appendix A, vol. I, Melbourne, 1917.

above a series of figures were chosen which seemed on all the evidence available to be the most reliable estimates, each for its time. These selected values were than fitted, giving the following equation:

$$y = 445.5 + \frac{1580.5}{1 + 5.342\, e^{-0.0243x}} \qquad \text{(xx)}$$

The calculated values are presented in table 192, and are compared with all the estimates in figure 122. The calculated point of inflection of the curve is the beginning of the year 1869.

TABLE 192

World

YEAR	POPULATION IN MILLIONS	YEAR	POPULATION IN MILLIONS
	Calculated		Calculated
Lower asympt.	445	1900	1520
1650	453	1910	1600
1700	471	1920	1671
1720	487	1930	1734
1740	512	1940	1787
1760	550	1950	1832
1780	609	1960	1870
1800	695	1970	1901
1810	750	1980	1926
1820	814	1990	1947
1830	887	2000	1963
1840	969	2020	1987
1850	1057	2040	2002
1860	1150	2060	2011
1870	1246	2080	2017
1880	1341	2100	2020
1890	1433	Upper asympt.	2026

The fit is obviously nothing to excite admiration. The most that can be said for it is that it strikes fairly through a flock of highly irregular points some of which are obviously poor guesses at world populations (judged to be poor not because of their divergence from the curve, but from comparison with nearby estimates in point of time). The marvel is not that the fit is bad, but that it is as good as it is, considering how world population estimates have to be made.

The calculated curve suggests that the world started on its present cycle of population growth somewhere in the sixteenth century, with a popu-

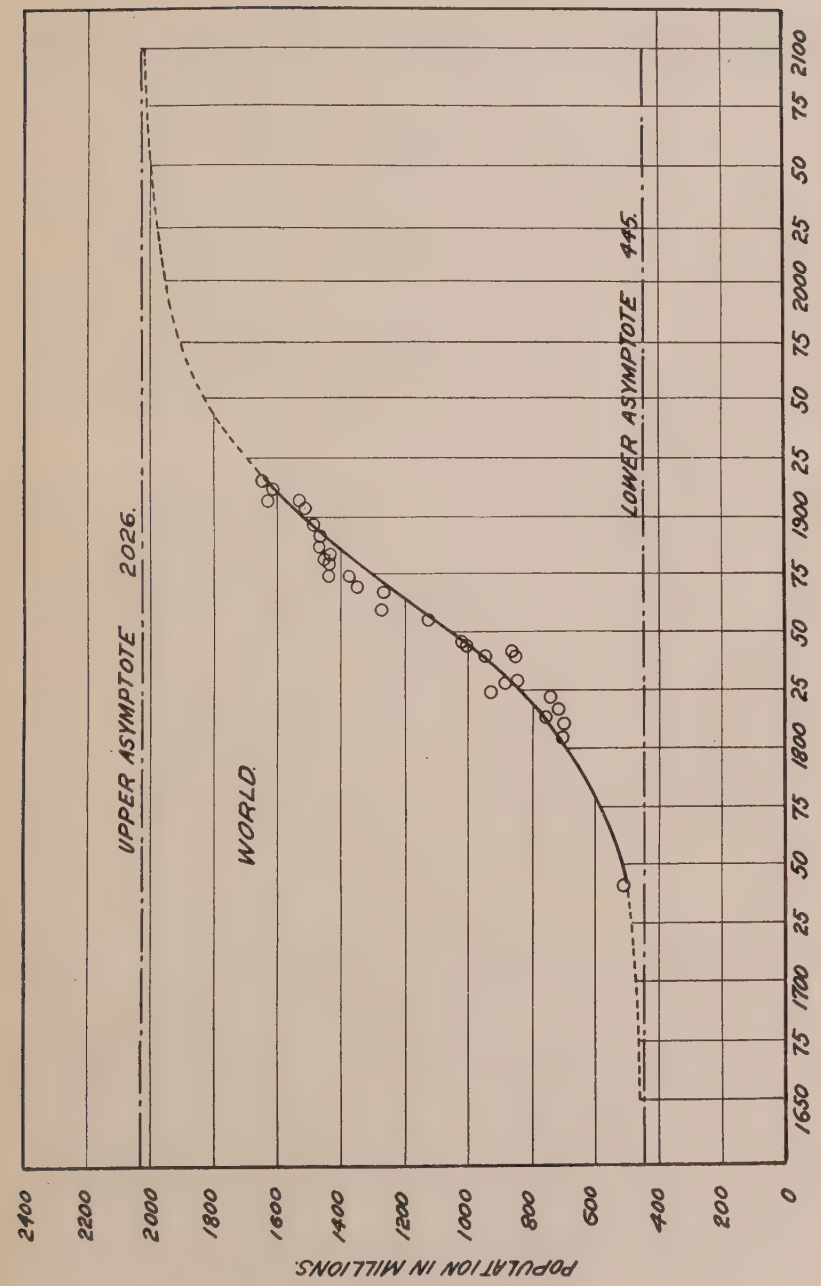

FIG. 122. THE POPULATION GROWTH OF THE WORLD

lation of probably between 400 and 450 millions. Up to 1869 the population of the world as a whole appears to have grown at an ever increasing rate. During the last fifty-five years the *rate* of growth has been declining. The predicted upper limit of population in this cycle of growth, which by 2100 we shall have come within 6 millions of reaching, is something over 2 billions. This does not appear to be a wild prediction, because we have already attained, according to the best estimates, a world population of over 1600 millions. Four more United States only have to be added to bring us approximately to the estimated upper asymptote.

Shall we then, or before that time, embark upon a new cycle, made possible and inaugurated by scientific discoveries which will free us from some or all of the inhibiting factors which limit food production by means at present known? Who can say?

THE POPULATION GROWTH OF A CITY

Since first publishing our population curve we have frequently been asked if it could be applied to smaller demographic units than whole countries. In particular, can it be applied to city growth, where in recent years industrial developments have caused enormous migration, increasing these urban populations at a much more rapid rate than excess of births over deaths would have done?

We have tried the curve with success on the population data of some of the largest cities in the world, and on a smaller, but rapidly growing city, Baltimore. Here we shall present only the data for Baltimore, referring the reader for a detailed discussion of this curve applied to the growth of New York City to an earlier paper.[8]

BALTIMORE

Baltimore city is an excellent case for the application of the population growth curve for two reasons. In the first place we have data on the population for a fairly long period (1800 to 1920, inclusive) without significant change in area, and in the second place Baltimore has had large waves of immigration at different times in its history, in connection with industrial developments. In dealing with city populations the alteration of the corporate areal limits makes a great difficulty. City fathers have a way of suddenly annexing large areas of surrounding territory and incorporating them within the city limits. This happened in Baltimore a few years ago.

[8] Pearl, R., and Reed, L. J., Predicted growth of population of New York and its environs, *Plan of New York and its Environs*, Publication no. 4, pp. 1–42, 1923.

But fortunately it is still possible to get for this city consistent figures for the time period mentioned above on the population of what is locally called the "old area." It is these figures which are used in the present discussion.

The theoretical curve has the equation:

$$y = \frac{0.805}{1 + 28.812\, e^{-0.0389x}} \qquad \text{(xxi)}$$

This curve had its point of inflection about July 1, 1886. Its ordinates, as well as the observed populations are presented in table 193, and figure 123.

TABLE 193

*Baltimore (Old Area).**

YEAR	POPULATION IN MILLIONS		YEAR	POPULATION IN MILLIONS	
	Calculated	Observed		Calculated	Observed
Lower asympt.	0.000		1900	0.506	0.509
1760	0.006		1910	0.575	0.558
1780	0.013		1920	0.633	0.636
1800	0.027	0.027	1930	0.680	
1810	0.039	0.047	1940	0.716	
1820	0.057	0.063	1950	0.742	
1830	0.081	0.081	1960	0.761	
1840	0.114	0.102	1970	0.775	
1850	0.157	0.169	1980	0.784	
1860	0.212	0.212	1990	0.791	
1870	0.278	0.267	2000	0.795	
1880	0.352	0.332	Upper asympt.	0.805	
1890	0.430	0.434			

* Data from United States Census reports.

It is seen that the graduation is satisfactory, and that the growth of population of this city has followed the same curve as the larger demographic units dealt with earlier in this chapter. The curve starts with a lower asymptote at zero, which is proper, because there is no evidence that as yet Baltimore has had more than one cycle of population growth, namely the one in which it now is.

The upper asymptotic population of 805,000 must be remembered to be of the old area of the city and exclusive of the recent annexations of area and population. As such it seems reasonable to those who know the city, and with the restrictive assumptions stated at the beginning of this chapter as applying to all population predictions.

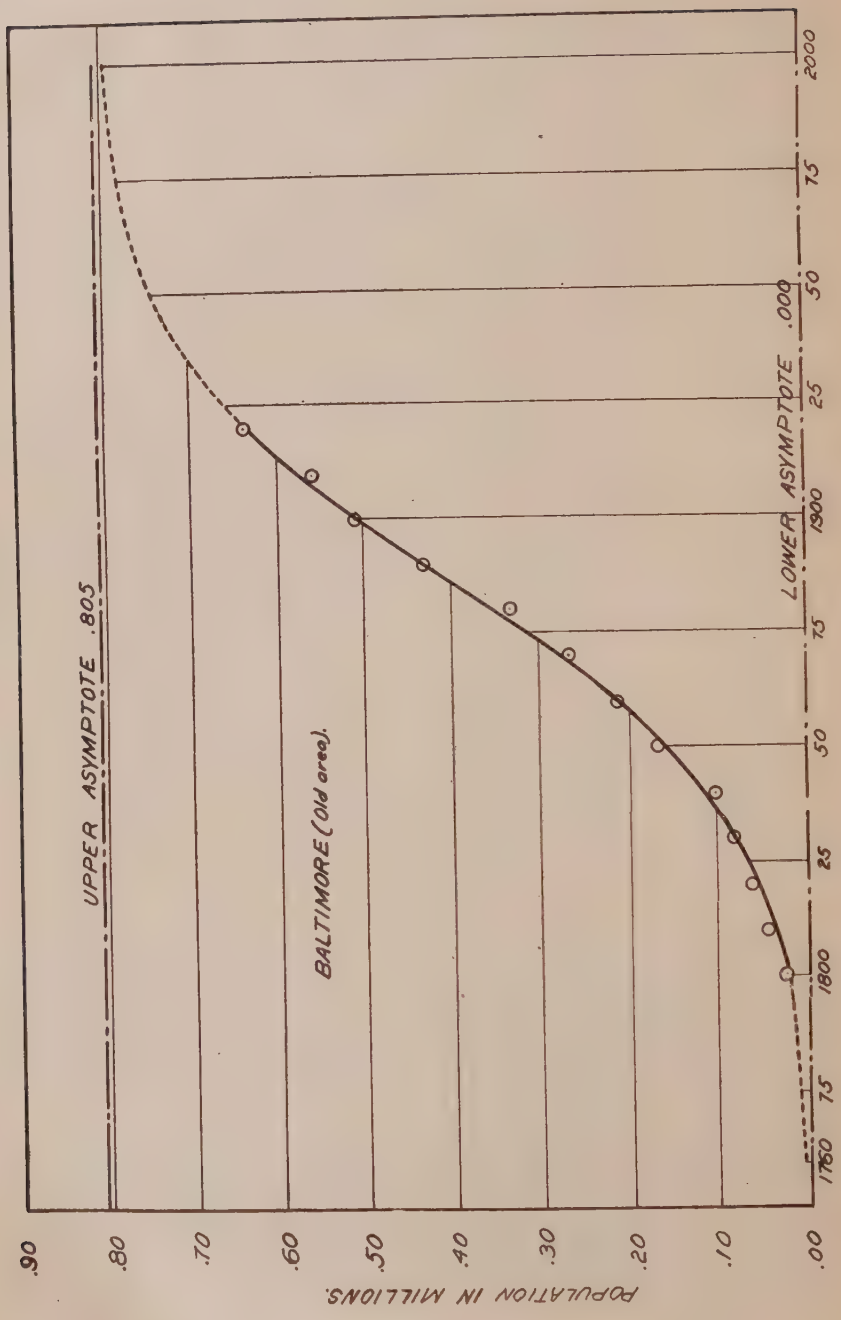

FIG. 123. THE POPULATION GROWTH OF BALTIMORE (OLD AREA)

CONCLUSION

In this chapter there has been presented evidence that the theoretical curve of population growth discussed in Chapter XXIV describes adequately the known phenomena in this field. This evidence consists in showing, for 16 different countries, the world as a whole, and one city (another, New York, by reference to material published elsewhere), that the curve does in fact describe the known (or, in case of the world, estimated) population growth with great precision and fidelity. The demographic units furnishing this evidence encompass a great range of diversity in cultural conditions, racial composition, industrial development, etc. We think that this evidence makes it probable that the curve is at least a first approximation to a descriptive law of population growth.

INDEX

A

Abdominal tuberculosis, 155, 156, 173.
Ability to function, 37–38.
Abscess, 150, 153, 160, 176.
Accidental deaths, 160, 161, 172, 176, 222, 300, 327.
Activity, 298–354.
Acute diseases, 21–22.
  miliary tuberculosis, 148, 149, 173.
  yellow atrophy of liver, 155, 175.
Adaptation, 169.
Addison's disease, 160, 167, 174.
Age, 26, 41, 49, 50, 51, 52, 54–57, 59–66, 83–84, 85, 92, 234–244, 262, 263, 267, 269, 292, 293, 325–342, 344–351, 355–371, 433–440, 475, 477, 484, 488, 508–511.
  index, 433–440, 475, 477, 484, 488.
  mean at death, 132–140.
  of parent, 110, 188.
Agricultural laborers, 338.
Agriculture, 531–533, 592.
  Department, U. S., 385.
Air, 164, 170.
Akron, Ohio, 428.
Albany, N. Y., 434, 436, 443, 444, 449.
Albuminuria, 155, 157, 176.
Alcoholism, 143, 158, 159, 174.
Alimentary tract, diseases, 144, 154–157, 162, 163, 164–168, 171, 172 175, 224–227.
Alleman, A., 179.
Alpine race, 29, 32.
Altbayerisch, 82.
Alternative, probability of, 108.
Alveolar angle, 53.
Amalgamation, racial, 177–180, 182–205, 229, 248–251.
Americans, 177–272, 294.
American Geographical Society, 6.
  Philosophical Society, 6.
  Public Health Association, 512.
  Society of Naturalists, 545.
  Statistical Association, 6.
Americanization, 180–205, 209, 210, 249, 251.
Amerind, 204.
Amputations, 176.
Ancestry, 285–290.
Anemia, 145, 147, 174.
Aneurism, 174, 353.
Angina pectoris, 145, 174.
Ankylostomiasis, 175, 514.
Anthrax, 145–147, 173, 301, 326, 327, 329, 330.
Anthropology, 5, 15, 31–32, 71, 85, 180, 247.
Anthropometry, 80, 180.
Apoplexy, 145–146, 167, 174.
  pulmonary, 148, 175.
Appendicitis, 155, 175.
Apples, 388, 390, 392, 404–407, 413–415.
Argentine, 106, 108.
Arithlog scale, 261, 443, 524, 551.
Armsby, H. P., 558.
Arnould, E., 353.
Arteries, diseases, 145, 148, 167, 174.
Arteritis, 146.
Arthritis, 153.
Assimilation, 182–205, 249, 272.
Assortative mating, 182–205, 280, 281.
Asthma, 148, 175, 328–330.
Asymptote, 566, 569, 570, 576, 581, 586–588, 590, 592, 593, 595, 596, 598, 600, 603, 605, 606, 609, 613, 615, 617, 619, 623, 625, 629, 634, 635.
Atheroma, 174.
Atlanta, Ga., 435, 436, 443, 444, 462.
Atrophy, 150, 154–156, 172, 176.
Atwater, W. O., 386.
Austrian, 186, 200–204, 207, 208, 211, 213, 214, 222–226, 294, 593–595.
Autocatalytic curve, 559, 570, 574, 581.

Autopsies, not random sample of population, 20–22.

B

Bacon, A. L., 358.
Bacteremia, 146.
Bacteria, 164, 170, 290, 291, 296.
Baker, G. W., 221–223, 295.
Baltimore, Md., 430, 443, 444, 449, 634–636.
Baltimore Health Department, 285, 357, 497.
Bananas, 388, 390, 392, 404–407, 412–415.
Barley, 529, 530.
Barrington, A., 158.
Bateson, W., 103.
Bauer, J., 355, 356, 359, 370.
Bavarians, 94.
  brain weight of, 18, 25, 26, 27, 29–32, 33, 34, 36, 40, 45, 49, 50, 51, 52, 53, 56, 57, 59, 64, 65, 66, 70, 72, 73, 74.
Beef, 389, 391, 393, 404–407, 411–415.
Beeton, M., 65, 359.
Bell, A. G., 359.
Bell, W. B., 515.
Belgium, 590, 595–597.
Beriberi, 155, 157, 173.
Bernhardi, F. A. J. v., 546.
Bertillon, J., 298.
Biological classification of causes of death, 143–176.
Biology, 5, 141, 166–171, 220, 223, 227, 238, 248, 254, 270, 379, 534–557.
Biometry, 5, 247.
Biostatistics, 178.
Birth control, 248.
  injuries, 149–152, 172, 176.
  premature, 149–152, 154, 165, 172, 176.
  rates, 207–210, 254, 268, 270, 272, 553, 554, 600.
  registration, 212, 245, 270, 271.
  registration area, United States, 184–186, 188, 189, 191, 200–203, 210–214, 228–232, 236–241, 244–246, 250–252, 255–259.
  statistics, 104, 182–213, 227–246, 251, 252.
  still, 109–110, 212–214, 251.
Birth/death ratio, 181, 227–246, 248, 252–272, 550–557.
Birthplace of parents, 184–186.
Bischoff, T. L. W. v., 17, 18, 23, 45, 49, 53.
Bladder, diseases of, 153, 167, 168, 175.
Blakeman, J., 59.
Blood and blood forming organs, diseases of, 144–148, 149, 153, 162, 163, 164, 171, 172.
Body size, 72, 87.
  weight, 49, 50, 53, 72–74, 87, 92.
Bohemian brain weight, 19–20, 23, 24, 25, 26, 27, 29–32, 33, 34, 36, 40, 49, 50, 51, 52, 53, 54–56, 59, 66, 71, 72, 75, 77, 78, 79, 81, 87.
Bones, diseases of, 142, 154, 176.
  measurements, 35, 53.
Boston, Mass., 430, 443, 444, 449, 455.
Bowman, I., 6.
Boyd, R., 17, 20, 83, 89.
Boyle, R., 585.
Brachycephaly, 81–82, 92.
Brain, diseases of, 145, 146, 152, 167.
  softening of, 145, 146, 167, 174.
  specific gravity of, 87.
  ventricles, 87.
Brain-weight, 15–92.
  Bavarian, 18, 25, 26, 27, 29–32, 33, 34, 36, 40, 45, 49, 50, 51, 52, 53, 56–57, 59, 64, 65, 66, 70, 72, 73, 74.
  and body weight, 72–74, 92.
  Bohemian, 19–20, 23, 24, 25, 26, 27, 29–32, 33, 34, 36, 40, 49, 50, 51, 52, 53, 54–56, 59, 66, 71, 72, 75, 77, 78, 79, 81, 87.
  correlation, 16, 33, 48–51, 57–58, 70, 73, 75, 80–81, 83–84, 85–86, 88–89, 92.
  data, 17–23.
  effect of age on, 22–23, 33, 52, 54–57, 59–66, 83–84, 85, 92.
  effect of disease on, 22, 39.
  effect of metabolic condition, 39.
  English, 20, 34, 53.
  French, 36, 53.

Brain-weight, Hessian, 17–18, 23, 25–32, 33, 34, 36, 40, 48, 50, 51, 52, 53, 54, 55, 59, 61, 62, 63, 64, 65, 66, 68, 69, 70, 71, 72, 83.
interracial differences in, 25–32.
means, 25–32, 40, 42, 43.
method of weighing, 18–19.
model series of data, 90.
"old" group, 91.
problems, 90.
and sex, 51–58, 87, 92.
sex ratio, 52–53.
and stature, 66–72, 83–84, 85, 89, 92.
Swedish, 19, 25–29, 33, 34, 36, 40, 46–47, 48, 50, 51, 52, 53, 54, 55, 57, 59, 61, 62, 63, 64, 65, 66, 67, 69, 71, 72, 83.
"total" series, 23, 49, 50, 52, 53, 55, 59, 61, 62, 65, 66, 67, 68, 69, 72, 73, 74.
types, 25–32, 91.
variation, 16, 23–26, 32–42, 53, 83–84, 85, 91.
"young" series, 23, 49, 50, 52, 53, 54, 55, 59, 62, 63, 66, 67, 68, 69, 77, 78, 79.
Branchial furrows, 168.
Bravais, A., 49.
Breast, 150, 152, 173, 176.
Breathing capacity, 36.
Breeding methods, 103.
Bright's disease, 22, 153, 165, 175, 222, 223, 485.
British, 186, 201–204, 207, 208, 211, 213, 214, 221–224, 249, 250, 294.
British Columbia, 292, 293.
Broca, 75.
Bronchitis, 148, 149, 165, 175.
Bronchopneumonia, 148, 175
Brown, J. W., 518.
Brown, L., 273.
Brownlee, J., 486.
Bryant, A. P., 386.
Buccal cavity, cancer, 155, 166, 168, 173.
Buffalo, N. Y., 437.
Bulloch, W., 278, 296.
Burger, M. H., 254, 550.

C

Calculi, 153, 155, 175.
California, 192, 193, 206, 256.
Calory, 385, 387–399, 401–403, 407–409, 411–414, 418–421, 592.
Calver, H. N., 6.
Cambridge, Mass., 437, 455.
Canadian, 181, 186, 192, 199, 201–204, 207, 208, 211, 213, 214, 224, 292, 293.
Cancer, 143, 150, 152, 155, 160, 162, 165, 166–168, 173–174, 176, 222, 223, 355, 356, 358–360, 364, 365, 370, 371, 486, 491.
Cannon, W. B., 542, 543.
Capacity, breathing, 36.
Carbohydrate, 387–397, 399, 401, 402, 406, 410, 412, 417–419.
Carey, B. W., 461.
Carpenters, 315.
Case fatality rate, 503–505.
Cattell, J. McK., 6.
Causes of death, biological classification, 143–176.
International Classification of, 142, 143, 145–150, 152, 154, 156–158, 171–176.
Census Bureau, 161, 184, 212, 221, 246, 442, 453, 487.
Cephalic index, 35, 36, 37, 53, 82, 180.
Ceratophyllum, 561.
Cereals, 388, 390, 392, 404–407, 413–415.
Cerebellum, 84.
Cerebral hemorrhage 145–146, 167, 174,
Cerebrum, weight of, 83–84, 89, 92.
Chancre, 150.
Chapin, C. V., 513.
Characteristic equations, 27, 54–57, 63, 64, 69–71, 73, 78.
Chemistry, 534, 535.
Chest girth, 53.
Child welfare, 165.
Childbirth, 150, 176.
Chinese, 183, 272, 352, 353, 547.
Chlorosis, 145, 147, 174.
Chocolate, 388, 390, 392, 404–407, 413, 414.
Cholera, 155, 173.

Chorea, 159, 174.
Chronic diseases, 21–22.
Chyluria, 153, 175.
Circulatory system, diseases of, 142, 144–148, 152, 162, 163, 164, 167, 171, 172, 174.
Cirrhosis of liver, 155, 175.
Civil War, 541, 546, 548.
Civilization, measure of, 227, 228.
Clendinning, J., 34.
Cleveland, O., 437, 449, 455.
Climate, 111, 295, 483.
Clothing, 170.
Cocoa, 388, 390, 392, 404–407, 413, 414.
Collis, E. L., 298.
Columbus, Ohio, 432.
Commerce Department, U. S., 385.
Commonwealth Fund, 282, 356.
Communicable diseases, 224–227.
Comparative anatomy, 168, 169.
Congenital debility, 150, 154–156, 165, 172, 176, 224–226.
  malformation, 123–131, 142–143, 147, 155, 156, 172, 176, 224–226.
  malformation of heart, 145, 147, 156, 172, 176.
Congestion, pulmonary, 148, 175.
Connecticut, 193, 196, 198–200, 206, 211, 215, 229, 230, 233, 235, 241–243, 251, 255, 256, 267.
Constitution, 170, 171, 220, 223, 227, 273–297, 301, 355, 379.
Contact, 274–277, 285–290.
Contingency, 95–98, 100, 187.
Control, 358–360.
Convulsions, 155, 158, 159, 174, 176.
Corn, 388, 390, 392, 404–407, 412–414, 529, 530.
Correlation, 16, 33, 48–51, 57–58, 64, 65, 70, 73, 75, 76, 80, 81, 83–84, 85–86, 88–89, 92, 93–102, 279–281, 359, 438–440, 473–496, 583.
  multiple, 88, 473–496, 518, 519.
  ratio, 58.
Costermongers, 323.
Craniometry, 32.
Criterion, 42, 43, 44–45, 180–182, 250.
Crossing and sex ratio, 103–113
Croup, 148, 149, 173.
Cubit length, 36.
Cultural epoch, 568, 575, 587, 588, 606, 623.
Cumming, H. S., 6.
Cummins, S. L., 297.
Curtis, M. R., 561.
Cycle of population growth, 568, 575, 576, 588, 589, 593, 595, 596, 598, 600, 602, 603, 605, 606, 613, 615, 621, 623, 632, 635.
Cysts, 150, 176.
Czechs, 19, 29.

D

Dairy products, 389, 391, 393, 398–407, 412–414.
Danish, 186, 211, 213, 214, 251, 596, 598, 599.
Darwin, C., 114, 169, 544.
Davenport, C. B., 103, 104, 537.
Davis, W. H., 184, 442.
Day, A. L., 6.
Dayan, 117.
Dayton, Ohio, 437, 443.
Deaf-mutism, 279.
Dean, A. S., 315.
Death, 180–182.
  causes, biological classification of, 143–176.
  causes, International Classification of, 142, 143, 145–150, 152, 154, 156–158, 171–176.
  mean age at, 132–140.
  rates, 134–135, 215–227, 251, 254, 268, 270, 271, 290–295, 325–342, 344–351, 373, 443–467, 484–487, 489–492, 516–518.
Debility, congenital, 150, 154–156, 165, 172, 176, 224–226.
Decorators, 315.
Delaware, 256.
Demography, 246, 477–484, 486, 488, 489.
De Morgan, A., 85.
Deniker, J., 29.
Density, 476, 480, 488, 518, 590, 595.

Dermal sensitivity, 36.
Destructiveness of epidemic, 487–495.
Deviation, mean square, 58.
Diabetes, 155, 156, 174.
Diabetes mellitus, 21.
Diarrhea, 155, 157, 164, 165, 175.
Dickson, J., 116.
Dictary studies, 418, 419.
Differentiation, 169, 540–543.
Digestive system, diseases of, 142, 147, 154–157, 162, 163, 164–168, 171, 172, 175, 224–227.
Diphtheria, 148, 149, 173, 519.
Disease terminology, 149.
Diseases, acute, 21–22.
of alimentary tract, 144, 154–157, 162–168, 171, 172, 175, 224–227.
of arteries, 145, 148, 167, 174.
of bladder, 153, 167, 168, 175.
of blood and blood forming organs, 144–149, 153, 162, 163, 164, 171, 172.
of bones, 142, 154, 176.
of brain, 145, 146, 152, 167.
chronic, 21–22.
of circulatory system, 142, 144–148, 152, 162–164, 167, 171, 172, 174.
communicable, 224–227.
of digestive system, 142, 147, 154–157, 162–168, 171, 172, 175, 224–227.
of endocrinal system, 144, 160, 162, 163, 165, 171, 172.
epidemic, 162, 173, 441–511.
of esophagus, 155, 175.
of excretory organs, 144, 153, 156, 157, 162–165, 167, 171, 172, 175.
of eye, 159, 174.
general, 142, 143, 146, 147, 152, 157, 162, 174.
of genito-urinary system, 142, 149–153.
of heart, 145, 148, 152, 174, 222, 223, 353, 442, 485, 487, 489–491, 495, 496.
ill-defined, 142, 161, 162, 172, 176.
of intestine, 155, 166, 168, 173, 175.
of joints, 154, 176.
of kidneys, 144, 153, 156, 157, 162, 163, 167, 171, 172, 175, 442, 485, 491.
of larynx, 148, 175.
of liver, 155, 156, 173, 175.
of locomotor organs, 142, 154, 176.
of lymphatics, 145, 147, 174.
of mouth, 155, 175.
of muscular system, 144, 153, 154, 162, 163, 165, 171, 172.
of nasal fossae, 148, 166, 168, 174.
of pharynx, 155, 175.
of prostate, 150, 175.
of respiratory system, 142, 144, 148, 149, 162–165, 167, 171, 172, 175, 224–227.
of sense organs, 142, 144, 157–159, 162, 163, 166, 171, 172.
of sex organs, 144, 149–154, 157, 162–165, 167, 171–173, 175, 176.
of skeletal system, 144, 153–154, 162, 163, 165, 167, 171, 172.
of spinal cord, 159, 174.
of spleen, 145, 147, 175.
of stomach, 155, 173, 175.
of thyroid, 148, 160, 168, 175.
of urethra, 150, 153, 167, 168, 175.
of uterus, 150, 175, 176.
of veins, 145, 174, 353.
Disseminated tuberculosis, 155, 156, 173.
District of Columbia, 193, 211, 212, 230, 233, 235, 241–243, 255, 256, 268, 270, 271.
Divorçe, 205.
Doering, C. R., 372.
Dolichocephaly, 81–82, 92.
Donaldson, H. H., 20, 72, 83, 87, 88, 562.
Drachsler, J., 183.
Drosophila melanogaster, 373–379, 585.
Dublin, L. I., 221–223, 295, 514, 515.
Du Pasquier, L. G., 569.
Duration of life, 65, 133, 135–138, 298.
Dysentery, 155, 173.

E

Earp, J. R., 315.
Ears, diseases, 159, 174.
East, E. M., 532, 533, 592.
Eastman, P. R., 207–210, 223–227.
Economic evaluation of public health activities, 514, 515.
Ectoderm, 166–171.
Eggs, 384, 389, 391, 393, 399–403, 404–407, 410, 412–414.

Elderton, E. M., 553.
Elderton, W. P., 47.
Elimination of unfavorable variations, 131.
Ellis, H., 124.
Embolism, 145, 146, 147, 174.
Embryology, 166–170.
Emotions, 542, 543.
Emphysema, pulmonary, 148, 175.
Encephalitis, 159, 174, 502–511.
Endarteritis, 147.
Endocarditis, 39, 145, 174.
Endocrinal system, diseases of, 144, 160, 162, 163, 165, 171, 172.
Endoderm, 166–171.
Energy, expenditure 298–354.
England, 144–146, 148, 150, 151, 153–155, 157, 159–165, 168–171, 254, 290, 291, 299–352, 396, 398, 441, 539, 550–557, 598, 600, 601.
English, brain weight, 20.
Enteritis, 155, 157, 164, 165, 175.
Environment, 164, 169–171, 219, 220, 229, 245, 274, 276–279, 295, 298, 300, 339, 379, 477–484, 486, 488, 489, 536.
Epidemic diseases, 162, 173, 441–511.
  encephalitis, 502–511.
Epidemicity index, 467–473.
Epidemiology, 441–496.
Epilepsy, 159, 174.
Epithelium, 164, 167, 168.
Equality of opportunity, 177.
Equations, characteristic, 27.
Erysipelas, 160, 173.
Esophagus, diseases of, 155, 175.
Etiology, 273–297.
Eugenics, 247.
Eupagurus Prideauxi, 130.
Evolution, 86, 123, 141–176, 177, 238, 248, 536–539, 543–549.
Excess Mortality, 453, 465, 466, 471, 476, 487–495.
Excretory organs, diseases of, 144, 153, 156, 157, 162, 163, 164, 165, 167, 171, 172, 175.
Exophthalmic goiter, 160, 168, 174.
Explosiveness of epidemic, 467–471, 473–487, 490–495.
External causes of death, 142, 157, 158, 162, 172, 176.
Extrapolation, 179, 253, 560, 561, 565, 586–588, 593, 595, 596, 598, 600, 603, 605, 606, 609, 611, 613, 615, 617, 619, 627.
Eye color, 180, 279.
  diseases, 159, 174.
Eyerich, G., 93, 95, 99.

F

Family history records, 282, 356–358.
Farmers, 338.
Farr, W., 353.
Fat, 387–400, 405, 409, 410, 416–419.
Fawcett, C. D., 42, 44, 46, 75, 76, 81, 99.
Fay, E. A., 279.
Feldman, A., 117, 118.
Femur length, 36.
Fertility, 206–210, 251, 272, 284.
Fertilization, time of, 119.
Fetus, 151.
Field, J. A., 551.
Finger length, 36.
Fish, 384, 389, 391, 393, 399–407, 412–414.
Fishberg, M., 115, 116, 179, 273.
Fisher, A., 516.
Fisher, I., 443, 551.
Flour, 384.
Food, 164, 170, 383–432, 528–533, 592.
Food Administration, U. S., 385, 396, 404, 412, 414, 415, 422, 423.
Foot length, 36.
Forearm length, 36, 279.
Foreign colony, 196, 199.
Foreign-born, 185–245, 248–252, 255–268, 272.
Fourfold tables, 99–100.
Franco-Prussian War, 603, 606.
Frankel, L. K., 515.
Franklin, B., 587.
Freeman, A. W., 513.
French, 36, 229, 250, 602–605.
Frequency distribution, 42–47.
Frost, W. H., 475.

Fruits, 388, 390, 392, 399–407, 413–415.
Fumigation, 513.
Furuncle, 160, 176.
Fusion percentage, 191–200, 250, 251.
racial, 182–205, 229, 248–251.

G

Galton, F., 279, 537, 559.
Galveston, Texas, 426.
Gamekeepers, 323.
Gangrene, 145, 147, 148, 175, 176.
Garbage, 417–432.
Gardeners, 324.
Gastro-intestinal diseases, 224–227.
Geddes, P., 110, 112.
General diseases, 142, 143, 146, 147, 152, 157, 162, 174.
hospital population, 21–22, 36.
paralysis of insane, 159, 174.
Genetics, 249.
Genito-urinary system, diseases of, 142, 149–153.
Germ layers, 166–170.
plasm, 537, 547–549
Germans, 186, 201–203, 207, 208, 211, 213, 214, 221–224, 250, 251, 294, 541, 544, 545, 605–610.
Giddings, F. H., 179.
Gini, C., 6.
Gladstone, R. J., 16.
Glanders, 145, 147, 173.
Glover, J. W., 373, 433, 434.
Goiter, 143, 160, 168, 174.
Gonococcus infection, 143, 150, 152, 153, 173.
Gonzalez, B. M., 373.
Goodness of fit, 47, 505, 506, 508–510.
Goring, C. B., 279.
Gout, 153–154, 174.
Grains, 388, 390, 392, 397–403.
Grand Rapids, Michigan, 443, 453, 455.
Graziers, 338.
Grease recovery, 422, 423, 431, 432.
Green, R. W., 6.
Greenman, M. J., 6.
Greenwood, M., 21, 22, 37, 49, 108, 278, 296, 298, 518.
Grouping, units, 20, 24, 30–31.
Growth, 65, 476, 483, 484, 488.
Guilfoy, W. H., 227.

H

Haeckel, E. H., 544.
Hair color, 180.
Hairdressers, 345.
Halsted, W. S., Dedication.
Hatai, S., 562.
Hatmakers, 345.
Hawkers, 323.
Hawley, P. R., 298, 304, 315.
Head breadth, 37.
circumference, 94, 96–97.
index, 53.
length, 37.
size and intelligence, 93–102.
Heart, diseases of, 145, 148, 152, 174, 222, 223, 353, 442, 485, 487, 489–491, 495, 496.
weight, 36.
Height, sitting, 53.
Hemorrhage, 145, 150, 174, 175, 176, 275, 276.
cerebral, 145–146, 167, 174.
Hemorrhoids, 174.
Henderson, L. J., 538.
Henry, W. A., 386.
Heredity, 171, 277–290, 298, 355, 356, 537.
Hernia, 155, 175.
Heron, D., 279.
Hersch, L., 517, 518.
Hessian Brain weight, 17–18, 23, 25–32, 33, 34, 36, 40, 48, 50, 51, 52, 53, 54, 55, 59, 61, 62, 63, 64, 65, 66, 68, 69, 70, 71, 72, 83.
Heterogeneity, 85.
Hill, J. A., 209.
Histogram, 46.
Historiometry, 535.
Hitchcock, J. S., 461.
Hoffman, F. L., 166.
Homicide, 160, 161.
Homogeneity, 32–34, 90, 91, 228.
Hookworm, 175, 514.
Hottentots, 541.

Housing, 111.
Howard, W. T., 141, 315.
Howe, H. E., 6.
Humerus length, 36.
Hungarians, 186, 201–203, 207, 208, 211, 213, 214, 222–226, 251, 294, 609, 611, 612.
Huntington, E., 495, 496.
Hybrids, 103–104.
Hydatid tumor of liver, 155, 175.
Hydrocephalus, 147, 156, 159, 172, 176.
Hygiene, 165, 218, 227, 245, 441, 442, 512–519.

I

Icterus, 150.
Ill-defined diseases, 142, 161, 162, 172, 176.
Illegitimates, 133–139, 182, 208, 210–212, 251.
Immigration, 207, 209, 210, 215, 217, 219, 223, 248, 249, 253, 272.
Immunology, 164.
Inbreeding, 103–104.
India, 546.
Indiana, 193, 211, 212, 230, 233–235, 241–243, 256.
Indianapolis, Ind., 437, 443.
Indoor occupations, 316–320, 324, 325, 331–336, 340–348.
Inebriates, 158.
Infancy, 142, 150, 162, 172, 176.
Infant mortality, 128–129, 132–140, 215, 217, 223–227, 513, 518.
Infection, 273, 274, 280, 281.
  purulent, 145, 147, 148, 173.
Inflection, point of, 568, 570, 573, 574, 589, 590, 593, 598, 602, 605, 611, 613, 615, 619, 623, 627, 629, 634, 635.
Influenza, 148, 149, 173, 229, 244–246, 252, 255, 262, 267, 441–501, 550, 552.
Inheritance, 171, 277–290, 298, 355, 356, 537.
Injuries at birth, 149–152, 172, 176.
Insanity, 279.
Insular sclerosis, 21.
Insurance agents, 315.
Intellectual capacity, 15, 81, 88, 89, 92, 93–102.
Intelligence, 218, 223.
  and nutrition, 102.
  and size of head, 93–102.
Interest, 538.
International Classification, causes of death, 142, 143, 145–150, 152, 154, 156–158, 171–176.
International Health Board, 270, 515.
Intestinal obstruction, 155, 175.
Intestine, diseases of, 155, 166, 168, 173, 175.
Iowa, 438–440.
Irish, 186, 201 202, 204, 207, 208, 211, 213, 214, 221–224, 250, 294, 295.
Italians, 106, 108, 186, 200–203, 207, 208, 211, 213, 214, 222–226, 250. 294, 295, 611, 613, 614.

J

Jacobs, J., 115.
Japan, 589, 621, 623–626.
Java, 627, 628.
Jews, 114–119, 181, 182, 199, 200, 204, 249, 250.
Johns Hopkins Hospital, 357.
Joiners, 315.
Joints, diseases of, 154, 176.
Juvenile Court, 285, 357.

K

Kansas, 193, 206, 211, 230, 233–235, 241–243, 256.
Keenness of sight, 36.
Kellogg, V., 544.
Kelly, F. C., 178.
Kentucky, 193, 206, 211, 230, 233–235, 241–243, 256, 268.
Kepler, J., 585.
Kerti, 356.
Kidder, A. V., 6.
Kidney diseases, 144, 153, 156, 157, 162, 163, 167, 171, 172, 175, 442, 485, 491.
  weight, 36.
King, G., 600, 602.
Kinship, 283, 284.
Kirkpatrick, R. E., 6.

Knibbs, G. H., 523, 589, 631.
Koch, R., 290.
Kopf, E. W., 353.
Krause, A. K., 6.
Kuczynski, 209.

L

Labor, accidents, 150, 176.
Lack of care, 162, 176.
Lagneau, G.-S., 115, 116.
Larynx, diseases of, 148, 175.
Latitude, 476, 480–484, 488, 491.
Leach, A. E., 386.
Lead poisoning, 155, 157, 174, 301, 326–330.
LeBlanc, T. J., 433.
Lee, A., 37, 49, 58, 75, 76, 81, 88, 94, 279.
Legitimates, 133-139, 182, 208.
Legumes, 388, 390, 392, 404–407, 413, 414.
Lehfeldt, R. A., 570.
Leprosy, 159, 173.
Leukemia, 145, 147, 174.
Levy, E. C., 513.
Lewenz, M. A., 88, 94.
Lewis, P. A., 281.
Life, duration of, 65, 133, 135–138, 298.
  tables, 373–379.
Lillie, F. R., 6.
Little, C. C., 113.
Liver diseases, 155, 156, 173, 175.
  weight, 36.
Locomotor ataxia, 152, 159, 174.
  organs, diseases, 142, 154, 176.
Loeb, J., 373.
Loewenfeld, L., 93, 95, 99.
Logarithmic curve, 561–567.
  scale, 261, 443, 524, 551.
London, England, 518.
Long bones, 36, 53.
Longevity, 355–371.
Longitude, 476, 480, 482, 483, 488.
Longstaff, G. B., 247.
Los Angeles, Cal., 455.
Lotka, A. J., 315.
Louisville, Ky., 449.
Lungs, 164, 327, 329.
  gangrene, 148, 175.
  tuberculosis, 148, 149, 165, 173, 222, 223, 273–297, 345, 353, 355–371, 485, 486, 489, 490, 496–501, 514, 515, 517,518.
Lymphangitis, 174.
Lymphatics, diseases of, 145, 147, 174.

M

Macdonell, W. R., 34, 37, 42, 44, 46, 49, 75, 76, 80, 81, 82, 99.
Maine, 192, 193, 206, 230, 233, 235, 241–243, 255, 257, 263, 264.
Maine Agricultural Experiment Station, 562.
Malaria, 142, 145, 147, 173, 515.
Malformation, congenital, 123–131, 142–143, 147, 155, 156, 176, 224–226.
  congenital, of heart, 145, 147, 156, 176.
  intensity, 127–130.
Malthus, T. R., 247, 523, 524, 530, 550, 566.
Manchester, N. H., 426.
Mandible, distance between foramina mentalia, 36.
  height, 36.
Marasmus, 150, 154–156, 172, 176.
Marchand, F., 17, 18, 22, 23.
Marriage, effective, 183–205.
Marshall, J., 17, 20, 83, 85.
Maryland, 193, 206, 211, 212, 230, 233–235, 241–243, 245, 257, 268.
Massachusetts, 183, 193, 195, 196, 198–200, 206, 209, 215, 230, 233, 235, 241–243, 245, 251, 255, 257, 266, 267.
Matiegka, H., 17, 19, 20, 24, 27, 30, 31.
Maxwell, J. C., 585.
Mayo-Smith, R., 179.
Mean square deviation, 58.
Measles, 142, 145–146, 149, 173.
Meats, 384, 389, 391, 393, 397–406.
Median, 378.
Medulla, 84.
"Melting Pot," 182–205, 248.
Mencken, H. L., 178.
Mendel, G., 548.
Meningitis, 159, 174.
Mental alienation, 159, 174.

Mesoderm, 166–169.
Messengers, 323.
Metabolic condition, effect on brain-weight, 39.
effect on variation, 35–40.
Metabolism, 144, 153, 298.
Metchnikoff, E., 161.
Michaud, G., 179.
Michigan, 192, 193, 206, 211, 231, 233, 235, 241–243, 255, 257.
Middle West, 192, 233, 244.
Miliary fever, 145, 147, 173.
Milwaukee, Wisconsin, 443, 452, 455.
Miner, J. R., 6, 255.
Miners, 315.
Minneapolis, Minn., 443
Minnesota, 192, 193, 206, 211, 212, 215, 231, 233, 235, 241–243, 257, 261.
Miscegenation, 192, 193.
Mississippi, 257, 268.
Mixed matings, 189.
Mode, 42, 43.
Morbidity, 514, 515.
Morgan, T. H., 112.
Morrison, F. B., 386.
Mortality, 141–176, 215–227, 298–354, 372–379, 514.
Mouth, diseases of, 155, 175.
Mucous membrane, 164.
Mühlmann, M., 39.
Müller, R., 103.
Multiple births, 187, 188.
correlation, 88, 473–496, 518, 519.
Murlin, J. H., 417.
Muscular system, diseases of, 144, 153, 154, 162, 163, 165, 171, 172.
Mutton, 389, 391, 393, 404–407, 413–415.
Mycoses, 160, 173.

N

Nagel, 115.
Naqada, 44.
Nasal fossae, diseases of, 148, 166, 168, 174.
Nashville, Tenn., 423, 437.
National Academy of Sciences, 6.
National Tuberculosis Association, 282, 356.
Native born, 185–245, 248–252, 255–268, 272.
Natural history of disease, 290–293.
Natural selection, 37–40, 86, 87, 131, 536, 543–549.
Nebraska, 257.
Negro, 228, 229, 245, 246, 250, 252, 255–260, 268–272, 554.
Nephritis, acute, 153, 175, 276, 485.
Nephritis chronica, 39.
Nerve cells, 167.
Nervous system, 142, 144, 152, 157–159, 161, 162, 163, 164, 166–169, 171, 172, 174.
Netherlands, 590.
Neuralgia, 159, 174.
Neuritis, 159, 174.
New England, 192, 233, 234, 243, 252, 255, 263, 267.
New Hampshire, 192, 193, 199, 206, 231, 233, 235, 241–243, 251, 257, 261, 264, 265.
New Jersey, 258.
New Orleans, La., 449.
New York, 183, 192, 193, 196, 198–200, 206–212, 215, 221–227, 231, 234, 235, 241–243, 251, 252, 255, 258, 261, 294, 437, 502–511, 634.
Newsholme, A., 139.
Newton, I., 585.
Nichols, J. B., 115.
Niddah, 116–117.
Norfolk, Va., 423.
Normal curve, 42, 46–47, 92.
North Atlantic Tuberculosis Conference, 273.
North Carolina, 193, 206, 211, 212, 231, 234, 235, 241–243, 245, 258, 268.
Northrop, J. H., 373.
Norwegian, 186, 211, 213, 214, 251, 613, 615, 616.
Nose, 53.
Noyes, B., 375, 379.
Nurserymen, 324.
Nurture, 135.
Nutrition, 111.
and intelligence, 102.

Nuts, 388, 390, 392, 395, 399–407, 413–415.

O

Oakland, Cal., 455.
Oats, 529, 530.
Occupation, 220, 298–354.
Oestrus, 114.
Ogburn, W. F., 6.
Ohio, 193, 206, 211, 231, 234, 235, 241–243, 258.
Oils, 388, 390, 392, 395, 399–407, 410–414.
Old age, 142, 160, 161, 162, 176.
Oleomargarine, 389, 391, 393, 399–407, 411, 413.
Ontario, 208, 292, 293.
Oranges, 388, 390, 392, 404–407, 412–415.
Oregon, 192, 193, 199, 206, 211, 258.
Organ size, 38.
Organic diseases of heart, 145, 222, 223, 485, 487, 489–491, 495, 496.
  soundness, 157.
Organological classification, causes of death, 143–176.
Osborn, H. F., 169.
Outdoor occupations, 315, 320–325, 336–342, 348–352.
Outlying points, 69.
Ovarian tumor, 150, 176.
Overcrowding, 178, 179, 247–250.

P

Painters, 315.
Palate, 53.
Pancreas, 156.
Parabola, 561, 563, 564.
Paralysis, 159, 174.
Parasites, 155, 175.
Parents, age of, 110, 188, 355–371.
  birth place of, 186.
Paris, France, 517, 518.
Parker, S. L., 315, 372, 373.
Parshley, H. M., 114, 119.
Passaic, N. J., 428.
Peacock, T. B., 34.
Peak death rates, 449–460, 507.
Peak-time ratio, 471.
Pearl, M. DeW., 103.
Pearl, R., 99, 103, 114, 119, 128, 178, 220, 254, 298, 304, 315, 356, 358, 359, 372, 373, 433, 443, 476, 504, 550, 558, 561, 569, 579, 584, 585, 634.
Pearson, K., 6, 16, 23, 32, 34, 37, 42, 44, 45, 47, 49, 52, 53, 58, 65, 88, 94, 95, 97, 99, 101, 102, 106, 124–125, 130, 136, 158, 279, 280, 281, 290, 291, 359, 504, 505, 537, 559, 581, 583.
Pedigree, 274.
Pell, C. E., 228.
Pellagra, 155, 157, 173.
Pelvis, 151.
Pennsylvania, 193, 199, 206, 211, 215, 221–223, 231, 234, 235, 241–243, 251, 258, 261, 294, 295.
Pericarditis, 174.
Peritoneum, cancer of, 155, 166, 167, 168, 173.
Peritonitis, 155, 167, 168, 175.
Perrin, E., 561.
Pertussis, 148, 149, 173.
Pesquera, G., 273,
Petroff, S. A., 273.
Pharynx, diseases of, 155, 175.
Philadelphia, Pa., 428, 443, 453.
Philippines, 546, 629, 630.
Phipps Tuberculosis clinic, 357.
Phlebitis, 147, 174.
Phlegmasia, 150, 176.
Phthisis, 148, 149, 165, 173, 222, 223, 273–297, 345, 353, 355–371, 485, 486, 489, 490, 496–501, 514, 515 517, 518.
Physics, 534, 535.
Pioneer, 209, 220, 292, 293.
Pittsburgh, Pa., 455.
Placenta, 151.
Plague, 145, 147, 173.
Pleurisy, 148, 167, 168, 175.
Pneumonia, 148, 149, 165, 175, 222, 223, 442.
Pneumonia cruposa, 39.
Pointsmen, 323.
Poisoning, 155, 157, 174.

Polish, 186, 201–203, 208–211, 213, 214, 224–226, 251.
Pons, 84.
Pooler, B. F., 497.
Population, 413, 414, 416, 523–637.
  autopsies not random sample of, 20–22.
  curve, 178, 179, 247, 250, 253, 558–637.
  general hospital, 21–22, 36.
  problem, 178, 179, 247–250, 253, 523–637.
Pork, 389, 391, 393, 404–407, 410, 412–415.
Porters, 323.
Potatoes, 384, 388, 390, 392, 404–407, 412–414, 519, 529, 530.
Pott's disease, 154, 173.
Poultry, 389, 391, 393, 399–403, 404–407, 410, 412–414.
Poverty, 517, 518.
Powys, A. O., 64, 65.
Prediction, 177, 565.
Pregnancy, accidents, 150, 176.
Premature birth, 149–152, 154, 165, 172, 176, 224–226.
Primary foods, 383, 384, 387, 389, 391, 393–398.
Pritchett, A. S., 561, 563, 564.
Proales decipiens, 375–379.
Probability of alternative, 108.
Probable error, 44–45, 60, 64, 78, 98, 106–107, 129, 133, 218, 325, 516, 517, 581–583, 587, 588, 592, 605, 621.
Production, 524–533,
Profile angle, 53.
Propinquity, 194,
Prostate, diseases of, 150, 175.
Protein, 387–400, 404, 407–409, 417–420.
Providence, R. I., 437.
Prussian death rates, 134–135.
Psychology, 15, 88, 180.
Public Health, 165, 170, 221, 223, 268, 270, 272, 296, 512–519.
Puerperal albuminuria, 155, 157.
  hemorrhage, 150, 176.
  septicemia, 150, 152, 153, 176.
  state, 142, 149–153, 176.
Pulmonary congestion, 148, 175.
  emphysema, 148, 175.
  tuberculosis, 148, 149, 165, 173, 222, 223, 273–297, 345, 353, 355–371, 485, 486, 489, 490, 496–501, 514, 515, 517, 518.
Punnett, R. C., 111.
Pure matings, 189.
Purulent infection, 145, 147, 148, 173.

R

Rabies, 159, 173.
Race combination, 200–203.
  crossing and sex ratio, 103–113
  prejudice, 192.
  stocks, 177–272, 293–295.
  suicide, 272.
Radius length, 36.
Railways, 525, 528.
Railway guards, 323.
Random sample, 32, 60.
Range, 126.
Ranke, J., 34.
Rectum, cancer of, 155, 166, 168, 173.
Reed, H. S., 581, 582.
Reed, L. J., 178, 315, 476, 558, 569, 579, 584, 634.
Registration Area, United States, 144–145, 148, 150–155, 157, 159–165, 168–171.
Regression, 28, 54–57, 58–74, 76–84, 92, 481, 494, 495, 559.
  coefficient, 64, 70.
  equations, multiple, 72–74.
  inter-racial, 83.
  intra-racial, 83.
Reid, J., 34.
Relapsing fever, 145, 147, 173.
Reproduction, 180–182
Reproductive system, 149–153, 154, 157, 162, 163, 164, 165, 167, 171, 172, 173, 175, 176.
Respiratory system, diseases of, 142, 144, 148, 149, 162, 163, 164, 165, 167, 171, 172, 175, 224–227.
Retzius, A., 17, 19.
Rheumatism, 153, 154, 174, 176.
Rhode Island, 192, 206, 231, 233, 235, 241–243, 258, 261.

Rice, 388, 390, 392, 404–407, 412–415.
Richmond, Va., 462, 513.
Rickets, 154, 173.
Ripley, W. Z., 29, 179.
Rivers, W. C., 279.
Robertson, T. B., 559, 570.
Rodents, 515.
Ross, E. A., 352.
Rotifer, 375–379.
Rubin, M., 228.
Rural, 229–235, 239–246, 248, 252, 255–271.
Russell Sage Foundation, 282, 356.
Russians, 186, 200–204, 207, 208, 211, 213, 214, 222–226, 294, 535.
Rye, 388, 390, 392, 404–407, 412–415, 529, 530.

S

Sadler's law, 110.
St. Louis, Mo., 452, 455.
St. Paul, Minn., 449, 455.
Salaman, R. N. 114.
Salpingitis, 150, 176.
San Diego, Cal., 426.
San Francisco, Cal., 430, 449, 455.
São Paulo, Brazil, 144–146, 148, 150, 151, 153–155, 157, 159, 160, 162–165, 168–171.
Saskatchewan, 292, 293.
Saunders, E. R., 103.
Saunders, W. B., Company, 6.
Scarlet fever, 145–146, 149, 173.
Schultze, O., 103, 104.
Schuster, E., 130, 279.
Sclerema, 150.
Sclerosis, insular, 21
Scotland, 615, 617, 618,
Scurvy, 155, 157, 174.
Seasonal distribution, 505, 506, 555–557.
Secondary foods, 383, 384, 387, 389, 391, 393–398.
Seedsmen, 324.
Segregation, 199.
Selected sample, 33.
Selection, direct, 86.
  indirect, 71–72, 81, 86, 92.
  natural, 37–40, 86, 87, 131, 536, 543–549.
Selective recruiting of occupations, 301–303.
Self-preservation, 158.
Senescence, 65, 332.
Senility, 142, 160, 161, 162, 176.
Sense organs, diseases, 142, 144, 157–159, 162, 163, 166, 171, 172.
Separation, ritual, 116–117.
Septic scarlatina, 146.
Septicemia, 145–149, 150, 152–153, 173, 176.
Servia, 617, 619, 620.
Sex, 507–511.
  determination, 103–113, 114–119.
  organs, diseases of, 144, 149–153, 154, 157, 162, 163, 164, 165, 167, 171, 172, 173, 175, 176.
Sex-ratio, 52–53, 103–113, 114–119, 476, 480, 488.
  abnormal, due to faulty registration, 115–116.
  among Jews, 114–119.
  excess of males, 107.
  higher for cross matings, 107–108.
Sheppard's correction, 25, 128.
Sight, keenness of, 36, 53.
Significant differences, 107.
Simon, C. E., 6.
Sims, 34.
Sitting height, 53.
Size of organ, 38.
Skeletal system, diseases of, 144, 153–154, 162, 163, 165, 167, 171, 172.
Skew growth curves, 574, 595.
  variation, 42.
Skewness, 42, 43, 44, 316.
Skin, 142, 144, 160, 162, 163, 164, 165, 169, 171, 172, 174, 176.
Skull, 169.
  auricular height, 37.
  breadth, 20, 26, 36, 37, 49, 50, 55–56, 75–82, 87.
  breadth to height index, 36.
  capacity, 34, 35, 36, 39, 53, 80.
  characters, 19.
  circumference, 37, 53, 80, 99.
  functions, 38.
  height, 37, 80, 87.

Skull, height to breadth index, 36.
length, 20, 26, 37, 49, 50, 55–56, 75–82, 87, 89.
ratio of height to length, 36, 37.
walls, 87.
Slavs, 32, 226, 250.
Smallpox, 160, 173.
Smith, H. F., 502–504.
Smith, W., 6.
Social classes, 342–352.
inheritance, 537, 538.
problems, 221, 247, 353.
status, 111.
Society of American peoples, 177.
Softening of brain, 145, 146, 167, 174.
South Carolina, 193, 206, 211, 259, 268.
Southern states, 192, 193, 234, 245, 260, 268–270.
Souza, G. H. deP., 159.
Spaeth, R. A., 315.
Span, 279.
Spanish, 106, 108, 250.
Specific death rates, 516, 517.
gravity of brain, 87.
Spencer, H., 169.
Spengarn, A., 146.
Spinal cord, diseases of, 159, 174.
Spitzka, E. A., 15.
Spleen, diseases of, 145, 147, 175.
weight, 36.
Springfield, Ill., 428.
Squeeze of hands, 53.
Standard deviation, 128–129, 316, 319, 320, 322, 468, 470.
of array, 63, 69, 73, 78, 80.
Standard of living, 209, 210, 219, 249, 531.
Statistical methods, 16, 48, 85–86, 128, 516–519.
Statistics of births, 104.
Stature, 26, 37, 41, 49, 50, 51, 53, 54–57, 64, 65, 66–72, 83–84, 85, 87, 89, 92, 279.
Stevenson, T. H. C., 518.
Stillbirths, 109–110, 212–214, 251.
Stomach, diseases of, 155, 173, 175.
Strength of pull, 36.
Stricture, urethral, 153.
Sugar, 388, 390, 392, 399–407, 412–415, 529, 530.
Suicide, 158, 159, 172, 176.
Sundbärg, G., 227, 228.
Survival, 545–547, 554.
Survivorship, 374, 376–378.
Swedish, 186, 211, 213, 214, 251, 619, 621, 622.
brain weight, 19, 25–29, 33, 34, 36, 40, 46–47, 48, 50, 51, 52, 53, 54, 55, 57, 59, 61, 62, 63, 64, 65, 66, 67, 69, 71, 72, 83.
Swiftness of blow, 36.
Sydenstricker, E., 475, 515.
Symington, J., 39.
Syphilis, 150, 152, 165, 173.

T

Taylor, G., 523.
Terre Haute, Ind., 426.
Tetanus, 142, 159, 173
Teutonic race, 29.
Thackeray, W. M., 85.
Thompson, W. S., 531, 532.
Thomson, J. A., 110, 112.
Thrombosis, 145, 146, 147, 174
Thury, M., 114, 116.
Thyroid, diseases, 148, 160, 168, 175.
Tibia length, 36.
Topinard, P., 85.
Touch, keenness of, 53.
Toxemia, 149, 157.
Trauma, 147.
Treitschke, H. v., 546.
Trybulowski, H., 6.
Tuberculosis, 142, 148, 149, 155, 162, 165, 173, 176, 222, 223, 273–297, 345, 353, 355–371, 485, 486, 489, 490, 496–501, 514, 515, 517, 518.
Tuberculous meningitis, 159, 173.
Tumor, 150, 155, 162, 174, 176.
Twins, 187.
Typhlitis, 155, 175.
Typhoid fever, 22, 155, 165, 173, 486, 491.
Typhus fever, 145, 147, 173.

U

Ulcer, 155, 175.
Uncinariasis, 175, 514.

United States, 144–145, 148, 150–155, 157, 159–165, 168–171, 177–272, 291, 383–432, 441–496, 502–504, 554, 561–567, 579–581, 589–592.
Units of grouping, 20, 24, 30–31.
Unoccupied, 303.
Urban, 230–235, 239–246, 248, 252, 255–271.
Urethra, diseases of, 150, 153, 167, 168, 175.
Urinary abscess, 150, 153, 175.
Urquhart, A. R., 279.
Utah, 192, 193, 206, 211, 231, 233–235, 241–243, 259.
Uterine hemorrhage, 175.
tumor, 150, 176.
Uterus, diseases of, 150, 175, 176.

V

Van Buren, G. H., 353.
Variation, 16, 23–26, 32–42, 53, 65, 75–76, 83–84, 85, 91, 99, 124–131, 136, 443–467, 470, 540–543, 581.
of sexes, 124–131.
skew, 42.
unfavorable, elimination of, 131.
Varices, 174, 353.
Vascular system, 145–148, 152, 162–164, 167, 171, 172, 174.
Vegetables, 388, 390, 392, 399–407, 413, 415.
Veins, diseases of, 145, 174, 353.
Ventricles of brain, 87.
Verhulst, P. F., 569.
Vermont, 193, 206, 232–235, 241–243, 252, 255, 259, 261, 263, 265, 266.
Vertebral column, 169.
Vertebrates, 168–169.
Violence, 222.
Virginia, 193, 206, 211, 232, 234, 235, 241–243, 245, 259, 268.
Viscera, 49.
Visceral weight, 35–37, 53.
Vital index, 181, 227–246, 248, 252–272, 550–557.
statistics, 104, 123, 141, 161, 440, 512–519, 560.
Vitality of peoples of America, 177–252.

W

War, 534–550.
Washington, 192, 193, 206, 211, 232, 233, 235, 241–243, 259.
Washington Academy of Sciences, 6, 534.
Waste, food, 417–432.
Wasting diseases, 22.
Watchmen, 323.
Water, 164, 170.
Weather, 495, 496.
Weber, H., 519.
Weight of brain, 15–92.
of cerebrum, 83–84, 89.
of heart, 36.
of kidneys, 36.
of liver, 36.
of spleen, 36.
visceral, 35–37.
Weigner, K., 86.
Weisbach, A., 51.
Weismann, A., 544.
Welch, W. H., 483.
Wernicke, J., 227.
West, 192.
Wheat, 384, 388, 390, 392, 404–407, 412–415, 529, 530.
White swellings, 154, 173.
Whitechapel skulls, 34, 82.
Whitney, J., 514.
Whooping cough, 148, 149, 173.
Wigmakers, 345.
Wilbur, C. L., 515.
Williams, 280.
Wilmington, Del., 428, 432.
Wilson, E. B., 6.
Wisconsin, 192, 193, 206, 211, 215, 232–235, 241–243, 259.
Woods, F. A., 535.
World population, 631–634.
Wright, S., 281.
Wynn, F. B., 37.

X

Xenia, 112.

Y

Yellow fever, 145, 147, 173.
Yule, G. U., 49, 78, 88, 473, 516

Z

Ziehen, Th., 15, 22.

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